Exhibit F

STATE OF MAINE
DEPARTMENT OF ENVIRONMENTAL PROTECTION

Public Hearing re
Proposed Chapter 584:
Surface Waters Toxics Control Program
Interim Statewide Criterion for Dioxin

Augusta Civic Center
6 November 1992

before:

OWEN STEVENS, Board Chair

OSMOND BONSEY, Board Member

GENE GENDRON, Board Member

CHRISTOPHER LIVESAY, Board Member

MARGARET ROY, Board Member

CHARLES STICKNEY, Board Member

CAROL TRACY, Board Member

Record of Proceedings
[Volume III]

prepared by Cyrille White
CONFERENCE REPORTING SERVICE
Thomaston ME 04861
16 November 1992

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DRAFT CHAPTER 584: Proposed Dioxin Limits
Presentation in Opposition by NRCM

Ron Kreisman, General Counsel, NRCM

My name is Ron Kreisman. I am the general counsel at the Natural Resources Council of Maine. It is good to finally have a chance to be before you. This issue has been played out in the press and we are anxious to have an opportunity to present our opinions on this to you. We anticipate that our presentation will take about three hours. We've talked with other people we've been able to contact over the past several weeks, including the Penobscot Indian Nation which will follow us, and it appears this schedule will be compatible with the needs of the rest of the audience, at least as we know it today. I am going to make an opening presentation, which I expect to last 20-30 minutes. Then we are going to present six testifiers on various topics which I will outline.

After the case you heard yesterday by the paper industry, I would imagine that you are honestly wondering and searching why anyone would oppose this rule. This is a tough case. I want to say that very clearly to you. The claims they made, at least on one level, are very reasonable. They've made good arguments. There are serious dollars at stake. We take those issues very seriously and have not entered this lightly. I want to share with you that since we learned at the end of July of the proposal by the Commissioner to go forward with this, we have--in a way that I frankly can't remember another matter that I've been involved in in my eight years in the Council--agonized over this and come back to it in many different ways. We have

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TESTIMONY OF OPPONENTS TO PROPOSED DIOXIN CRITERION

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gone through a hard soulsearching process as to what we should do on this, knowing the potential impacts to the mills, knowing how the Natural Resources Council at least would be labeled as unreasonable, as dogmatic, as impatient, as not sensitive to some very clear needs from the paper industry. We knew the resources that the industry would bring to this hearing, which you saw yesterday. Frankly, we knew of the tremendous political pressure to get this rule through that we've seen manifest itself in different ways.

So for all those reasons, we kept asking ourselves whether we could and should just walk away from this because, if this is really just an interim rule, if it would really just last two years, and if we would just wait and have a safe day now and for the next two years, there really isn't a lot of reason to be here and to spend an enormous amount of resources--your resources and everyone else's. And so to reach our decision to be here today we asked ourselves a series of questions and we kept re-asking these questions. In order to present our case to you and give you an outline of what we think the testimony will show, I want to take you through the kinds of questions we have been asking ourselves as to why we should be here.

First, we looked at the title of this rule as an interim rule and we asked ourselves: how long will this rule really last? More specifically, how long would the effects of this rule really last? What are we really talking about here? Will the effects of a 30 ppq end-of-the-pipe that you've heard about, or the 0.5 ppq in the water, really just be for two years, as you've heard the paper industry say; and then, whatever happens in the EPA re-assessment would go right in? Or will the rule really last for a lot longer than that, reasonably, based on the

calculation? You've heard me say in our August hearing that we think the effects of this rule will last at least five to seven years.

What I want to do to tell you how we thought about our first question is to walk you through my thinking on that, so you don't just hear this as a conclusory assertion but so you really understand, and hopefully agree, that when you are thinking about the impacts of this rule, you are not thinking in a two-year time context but in about a seven-year time context. You'll notice that the paper industry has been very careful not to walk you through the kind of analysis that I think is necessary.

You heard from Commissioner Marriott yesterday, you heard from EPA, that we are really talking about two sets of licenses; we are not just talking about a federal license, we are not just talking about a state license, we are talking about both. And so I've outlined, at Tab 1, how we think implementation of a 0.5 ppq standard would impact federal licenses and how it would impact state licenses. The top of the page talks about what happens if the Board adopts, for federal licenses, a 0.5 ppq standard--how long will it last? The answer that I am suggesting is that it will last at least until 1998 and probably until the next century. Here is why.

There are two ways that the federal government, under the National Pollution Discharge Elimination Permit System [NPDES] would incorporate this 0.5 into their licenses. First, if you adopt a 0.5 standard, they could issue new licenses to the mills. Because the mills have all appealed their licenses, EPA could pull those licenses right now and simply issue a new license. If they issued a new license at a 0.5 standard, the licenses would have a five-year

effect, until 1998. What I then go on to say--and I think it's a very important point--even if a new, more restrictive dioxin water quality standard were to be promulgated by EPA during the term of that license--in other words, before 1998--it is extremely difficult (and I think EPA will tell you the same thing; they've certainly said it to me) to re-open federal licenses without the agreement of the licensee. So essentially you have to presume you are going to be stuck with the number until 1998 and the effects of that in terms of discharges.

I then go on to note that, given delays in re-issuing federal licenses--frequently two years--we could see a post-re-assessment dioxin water quality standard not go in until the year 2000. And even if it was timely issued in 1998, there could be a compliance schedule, just like you see right now, for three years to come into compliance. I am not saying there would, but certainly no one has said there wouldn't--which would extend the period of time another three years when the 0.5 could be in effect. Then there is the possibility of litigation, which has happened in innumerable instances with the paper industry and the dioxin standard. Even if EPA chose instead to modify their existing permits, substituting 0.5, because licenses are under appeal right now, the appeal period would cause these licenses to start again. So therefore you are going to be on the exact same schedule as if a new license were to be issued in 1993. In sum, at the federal level, you are looking until 1998 with almost certainty and very likely, given the history of dioxin regulation, significantly beyond that.

But there are still state licenses. You have control of state licenses. Are we really on the same decade-long

schedule when we are talking about state licenses? For that, I'd like you to look at the next page under Tab 1. If the Board adopts 0.5, how long would it last under state licenses? Is it really the two-year period the paper industry is talking about?

The answer I suggest again is, at least until 1998, but for a different set of circumstances, and possibly until the next century. I provide two reasons for that. One is very matter-of-fact and I call it the "honest" answer: politically and practically, once the paper industry obtains federal licenses that last until at least 1998 with a 0.5 standard, we are kidding ourselves to think the state would do anything different. I just don't believe it is going to work that way and I think you'd be very hard-pressed to find a rationale to say that it would. But there is a more detailed answer.

If this rule is adopted, DEP will issue new kraft mill licenses, which all have either expired as of now or will expire early in 1993. They will issue new licenses with the 0.5 standard. They will give the mills a five-year schedule. Assume, though, that the language of the proposed dioxin rule is changed from what it is now to open it up more than is possible now. So you require that all new kraft mill licenses have re-openers in them, so that if a new dioxin standard is enacted by EPA, it can be inserted once the EPA re-assessment is completed and before the expiration of the license. Assume further the proposed rule is further changed to require DEP to present the BEP with a new proposed dioxin water quality standard based on the findings of the EPA re-assessment no later than six months (just to pick a date) after the completion of that re-assessment.

If you go through the timeline and you assume that the EPA re-assessment is out in September--which we are not sure of and the paper industry told you several times yesterday they don't think is even going to happen--BEP would not even have a new rule proposed to it until March of 1994. If the rulemaking is controversial, there are delays, and at best, under the most expedited schedule imaginable, the new standard is rolled into kraft mill licenses by the end of 1994, substituting for the 0.5. And then a three-year compliance schedule takes you until 1998. And then, if it is a restrictive standard, the paper industry will sue--as happened in 1990 in this state, as happened in Washington, as Dan Boxer just explained to you, to overturn the 0.013 there. So that will cause another eighteen months' delay. Realistically, again, if you adopt this standard, even under the most heightened schedule, you are looking at around the year 2000. That's how we answered the first question: how long will this rule practically last--looking beyond the words of a two-year interim rule?

We then moved on to the next question: what exactly does this rule say about the levels of dioxin that will be discharged during this five-seven-eight-year process, because if the levels aren't a significant problem, who really cares how long the rule will last? The rule really says two things and you got at it in your questioning of several people, including Commissioner Marriott. At best, under the rule, discharge levels will stay at the status quo. I say "at best" because the data that DEP submitted to you yesterday shows all the mills are discharging under the 30 ppq/0.5 standard. So what we know is that this rule was designed to essentially enshrine the status quo. That was the purpose of it. And so we can assume that, at best, what

will happen is that discharge levels will stay right there. That's why the paper industry likes the rule. That is the understanding of the Commissioner's office. It is to keep things where they are. It will not require any changes in processes to bring down dioxin levels.

If that's what the rule will do at best, what will it do at worst? Legally--and I think it is important to talk legally, if nothing else, because that's what I'm paid to do--if the mills use the rights that are afforded to them under the license, based on the data handed out by DEP yesterday, six of the seven mills could substantially increase over current levels. There is one mill at Lincoln where there appears to be only one data point from 1988. You heard testimony yesterday--and Mr. Bonsey elicited that testimony from several people--that said they wouldn't increase discharges because they are not going to play around with the system, it's not in their interest and things like that. That may be true, it may be false. Frankly, I haven't the technical background nor, respectfully, do I think any members of the Board have the technical background to know whether that would occur or not. But as a legal rule-setting situation, you are granting them the right to substantially increase their discharge. Commissioner Marriott yesterday said that the rule is not intended to allow any more discharge than the current level. I take him at his word; I know that isn't the intent. But legally it allows, at least for six out of the seven mills, a substantial possible increase.

The third question we asked ourselves: what are the dioxin levels currently in fish? In other words, if at best the rule enshrines the current discharge levels, shouldn't we be looking at the fish to see if it is a problem--looking

at the fish that people will be eating at these levels for the next five-seven-eight years? Obviously, if we concluded that the levels of dioxin in the fish were not a public health threat, the five-to-seven-year problem wouldn't concern us. So we focused on the levels in fish right now.

To do this, we looked at last year's fish data done by DEP in their monitoring program and we made best guesses as to what the levels would be in the future. Fortunately, last week and this week we were able to get the data that is coming out this year as to what the fish levels actually were. That information will be presented to you. In fact, you will note that in several areas the levels are not going down. The dioxin levels have actually gone up above fish advisory levels. With these calculations, we then knew what levels it was reasonable to expect would be in each fish that people would be eating.

We then looked at what the levels in the fish could be, if the legal limits were taken advantage of. We calculated the legal limits. We used a reasonable bioaccumulation factor. Then you can calculate what would be expected to be in the fish. Knowing what we expected to be in the fish and what could be in the fish, we turned to the potential health effects over the next five-to-seven years. We had to ask ourselves, first: how much fish are people eating or would they reasonably be expected to eat if the fish advisories were lifted and they were told that the waters were safe?

For that we contacted Dr. Barbara Knuth, whom we had retained after a nationwide search a year earlier to assess for us the ChemRisk study and what reasonable fish consumption rates were in Maine as compared to other states. You will hear her testimony. In our search to answer that question, we re-affirmed that significant segments of the

Maine population are eating fish fairly frequently, and that there were large statistical/methodological problems with the ChemRisk study. We also re-acquainted ourselves with the knowledge that when that study was done, Commissioner Marriott and Steve Groves asked for it to be peer-reviewed by two nationally known outside experts, one at EPA and one at the University of Michigan. Although it wasn't mentioned to you by the DEP yesterday, the results of that peer review were not heartening in terms of the adequacy of the ChemRisk study.

So knowing how much is in the fish, how much could be in the fish, and how much people were consuming, we were able to then ask the next question, really the ultimate question: is there a health concern over the next decade, or slightly less, from eating reasonable amounts of fish currently in the rivers or possibly under the proposed rule? Again, if the answer was negative, we would walk away from this hearing.

How did we go about answering that question? First we contacted people very much involved in the re-assessment in whom we had faith. These people were either authors of chapters for the re-assessment or they had been asked by EPA to review the re-assessment. We laid out the fish levels to these people and the time frame. What we heard back was disquieting. We heard back predominantly that, over five-to-seven years (not over a 70-year exposure period for cancer), subtle reproductive and developmental effects of dioxin could be expected to occur. These effects would be related to fish consumption over a short time frame, as I mentioned, and not over a 70-year exposure period. We learned about the increased risk that all modest fish consumers would bear from eating fish, even at current

levels and not a probabilistic formula of 1-in-100,000 or 1-in-1,000,000.

We also learned that when we were thinking about this whole subject we had to look at the background levels of dioxin that each of us bears in our bodies and that is certainly borne by modest fish consumers. Frankly, we were startled by what we learned about the background body burdens that each of us carries and the doses that each of us is taking in every day from foods, from other sources. We will present testimony on that. None of those assumptions about background body burdens that we all bear, or the doses of dioxin that we take in--not counting the fish--were discussed by the paper industry yesterday. In all the toxicological modeling that they did for you, the assumption was that the only source of dioxin that was going into your body, or was there, was coming in from the fish. That predicated all their theories on what kind of dose you are getting. A very different assumption, we think, reflects the real world.

Finally, we found out that there was a very different toxicological approach, besides the dose approach that you heard yesterday from the paper industry, which was actively being discussed. What we heard back from these people was confirmed by what we were reading from EPA. I would ask you to turn to Tab 2. You'll see an article from the Wall Street Journal of October 16, 1992, titled "Dioxin's Health Risk May Be Greater Than Believed, EPA Memo Indicates." That memo I will get to shortly. The second paragraph:

"Erich Bretthauer, an assistant agency administrator, told EPA chief William Reilly in a memo [a memo following the re-assessment you've heard about] that evidence indicates dioxin, a ubiquitous industrial byproduct, may

have reproductive, behavioral and immune-system effects on humans at concentrations close to levels in the general environment. Dioxin already has been linked to cancer. The panel, consisting primarily of academic scientists, has been looking at dioxin as part of the EPA's reassessment of the chemical."

We got that memo that went to Reilly from Erich Bretthauer. I would invite you to read it. It's not a memo from the popular press. I would invite you to turn to page 2, where there is a checkmark, where Mr. Bretthauer is talking about his interpretation of salient features of the discussion, including the fact that "Certain non-cancer effects, including changes in endocrine function associated with reproductive function in animals and humans, behavioral effects in offspring of exposed animals, and changes in immune function in animals have been demonstrated. Some data suggest that these effects may be occurring in people at body burden levels that can result from exposures at, or near, current background."

Finally, I have included an article in the Environment Reporter in which Bill Farland--the same Bill Farland that Dan Boxer quoted yesterday as saying workshop participants should refrain from predicting results--went on to talk about such things as "...scientists at recent meetings reported a host of non-carcinogenic effects at very low dose levels--near background levels--as well as the ability of dioxin to cause cancer in humans at high doses." On the next page, the article goes on: "Farland countered [countering an argument by someone at the National Council of Paper Industries] that the revised studies show that 'we have to be very cautious about any additions of dioxin to the environment. We must be very concerned about these

high background levels of dioxin and what they may mean for human health.'"

We were further disquieted by the continuing evidence which was not, as we found, scattered, anecdotal, and uneven, but furthering consensus about much more concerns about other impacts of dioxin also. The expert conclusion we were hearing was that modest consumption of dioxin-contaminated fish at current levels would, at best, perpetuate and, at worst, could exacerbate dioxin body levels that were already high enough to be a significant public health concern when looking at developmental and reproductive effects over the short term.

We also learned--and you heard it yesterday and there is no way to dance about it--that EPA, from a regulatory point of view, has completely abandoned the field for now to the states. It has made the situation for boards like yours and organizations like ours very difficult. There is no other way of saying it. EPA made a decision in January of 1990, before the re-assessment, that they would allow a range of values for the states. And they have held to that decision, notwithstanding what we think is fairly overwhelming scientific evidence to the contrary. I can only tell you that, in amendments to the Clean Water Act being sponsored by a huge consortium of municipal and environmental groups and others, there are major provisions to make EPA water quality standards mandatory on the states.

Finally, we asked ourselves the question I have to ask myself as a lawyer: what does the law say? what does the law say that you have to apply? That law is found at Tab 4. You will see a paragraph that says: "The board may substitute site-specific criteria or alternative statewide criteria for the criteria established in paragraph A

[paragraph A being EPA criteria] only upon a finding that the site-specific criteria or alternative statewide criteria are based on sound scientific rationale [and you've heard that phrase but you really didn't hear discussion yesterday of the other part of that test that has to be met] and are protective of the most sensitive designated uses of the water body, including, but not limited to, human consumption of fish..." You cannot enact a standard that you do not find to be protective of human consumption of fish.

That chain of reasoning is what brought us to you today and what will inform our discussion. We concluded that we need to keep that 0.013 in the federal licenses in order to frankly drive down further the levels of dioxin, notwithstanding the very substantial improvements the paper industry has made to date, for which they should be publicly applauded.

Before outlining specifically what our witnesses are going to say, I'd like to talk about three other items. First, what is happening in other states? You heard a lot of testimony yesterday and I'd like to give you a somewhat different slant on that. It is unquestionably true that there are a number of states that have adopted a weaker standard. It roughly breaks down as half and half, although I think slightly more states have adopted a weaker standard than 0.5. I'd like you to note where those states are located, for the most part: Alabama, Arkansas, Georgia, Louisiana, Mississippi, South Carolina, Tennessee, Texas, Virginia. They are states--without wanting to disparage anyone or any place--which Maine does not usually align itself with in terms of its environmental tradition. So certainly there are a number of states with a number of paper mills that have adopted a weaker standard, but there

are also a number of states that have not. For instance, Michigan, Minnesota, Montana, Oregon, Pennsylvania, Wisconsin. Idaho, from our understanding, had an EPA limit imposed on it. Finally, California.

I'd like to spend a couple of minutes talking about California and then what happened in North Carolina earlier this year. I would note that these states have faced exactly the same thing that you are facing. I know that from abundant articles in the popular press, from talking to colleagues in those states. You remember Dan Boxer yesterday saying we really shouldn't look at California because California has mills that just dump into the ocean. You have a huge amount of dilution, it's not a problem.

I'd like you to turn to Tab 3. What you are going to see is potentially the most interesting document in this array of documents that you will have seen over the last couple of days. That document is a joint letter from Louisiana-Pacific Corporation, located on the ocean in Samoa, California, and something called the Surfrider Foundation, which has a chief legal counsel by whom I am humbled, because the enforcement agreement that was negotiated between the Surfrider Foundation and Louisiana-Pacific Corporation, as indicated here, makes it "the first pulp mill in the United States to produce market kraft bleached pulp continuously without any chlorine chemistry. The production of absolutely chlorine-free pulp would eliminate the discharge of chlorinated dioxins and furans..." So, as for California having weaker standards, that is the result.

I'd like to also tell you about happened in North Carolina this spring. North Carolina is a state surrounded by states that have a weaker standard. From what I

understand, for the second time North Carolina was confronted by efforts like efforts here to weaken their water quality standard, acting as an outlyer in the South. North Carolina took testimony from, among others, Dr. Ellen Silbergeld, one of our witnesses here, took testimony from one of the principal EPA toxicologists involved in the re-assessment, Dr. Linda Birnbaum. And North Carolina decided not to change their water quality standard. They have five mills in that state.

The second point I'd like to talk about is that Dan Boxer made mention yesterday that he simply couldn't understand why everyone was objecting now to this rule-making when no one objected to the 30 ppq standard when it went into state licenses and lasted up until June 1993. I'd like to suggest a couple of simple answers, focusing on the fact that there were EPA permits at that time--that were issued, about to be issued, in effect, about to be in effect, in the offing--in which they were enforcing a 0.013 standard. With limited resources and with an EPA permit in place governing the playing field, there was no need to go after the state licenses and engender the type of proceeding that has now been engendered, if it could be avoided.

One last comment before talking about our presentation. You are going to hear from Dr. Claude Hughes, who could not resist talking about Dan Boxer's 55-gallon-drum analogy. We are so tired of that analogy as a way to describe and potentially dupe lay people as to the impacts of dioxin. I would urge you to listen very carefully to what he has to say about that.

Let me now talk about our presentation. Our presentation is going to follow the questions that I presented to you. First, Peter Washburn, staff scientist

at the Natural Resources Council, with a master's degree in environmental toxicology, deeply involved in the testing that has gone on on Maine rivers, is going to present to you data showing the levels of dioxin in Maine fish now. Following him, Dr. Peter deFur, senior scientist at the Environmental Defense Fund, will have a short discussion about bioaccumulation rates, which will then allow you and us to calculate what potentially could be in the fish if the legal limits allowed in this rule were fully utilized. Following that, Dr. Barbara Knuth, whom I've mentioned, will discuss fish consumption data. From those three, you will know how much is in the fish, how much could be in the fish, what are people expected to consume. Then Thomas Webster, researcher at the Center for the Study of Biological Systems at Queens College, will discuss with you the data that exists nationally and internationally on the levels of dioxin in our bodies and the dose we are taking in. Mr. Webster was part of the EPA review panel. Following that, we will be able to assess health impacts. Dr. Claude Hughes, principal reviewer of the reproduction and developmental part of the EPA re-assessment, both a medical doctor and a PhD in neuroendocrinology at Duke University, will discuss with you reproductive and developmental effects. Finally, Dr. Ellen Silbergeld, professor of toxicology at the University of Maryland, author of one of the sections of the dioxin re-assessment, dioxin researcher in her own right, will discuss further the reproductive aspects of this case and will go on to talk about others.

I want to stress, for the record, that Mr. Webster, Dr. Hughes, Dr. Silbergeld are not being paid as consultants for the Natural Resources Council of Maine, should that ever come up in their role in the EPA re-assessment. They are

simply taking out-of-pocket expenses to be here. Following their presentation, I would like to give a very short closing.

LIVESAY: One of your concerns with the limitation in a state license is that if a new federal standard were adopted there would be some significant time delay before that standard could then be incorporated in some sort of state license because of the need to hold hearings and that sort of thing.

KREISMAN: You mean a new dioxin re-assessment number? That's right.

LIVESAY: What is the problem with conditioning a state license so that, assuming there were a new federal dioxin standard within a certain range, then that standard would in fact be the standard applied in that license, period?

KREISMAN: Without going into the legal issues, I don't think it changes the timing, which is what I think you are getting at. Let me explain why. The re-assessment is not going to be completed, at best, until next September. The re-assessment is not going to produce a water quality standard. It is going to be the basic science from which EPA will then derive a water quality standard. EPA has been quoted that it will take "several years" further to develop that water quality standard. So if we really are on a two-to-three-year schedule for developing that, once that gets rolled into state licenses automatically, and once a compliance schedule is added to that, you are really talking

about five-six-seven years anyway, if you follow my timing on that.

LIVESAY: Then I am confused as to why anybody would call this an interim standard.

KREISMAN: I don't think it is an interim standard. That was the point of my discussion. Let's be clear. This rule doesn't sunset. All it requires is that, two years after the rule began or if EPA sets a water quality standard within a two-year period, the DEP has to come back before you. Now if DEP chooses not to come back before you, the Natural Resources Council has to go for a mandamus action in court. The point is that the rule has some fuzzy limits, but what will spin out from adopting 0.5 goes way beyond the time limits of that rule. That's the point I am trying to make.

LIVESAY: It just seems to me there ought to be some sort of mechanism that would enable anybody issuing a license under this rule to condition that license so that if there were some sort of new assessment--assuming that assessment is within a particular range--then whatever that standard might be would be the standard that would apply in the particular license.

KREISMAN: I am suggesting, without having given any thought to whether that is legally possible, even if that were possible, given the time frame we are operating under, to get that new water quality standard--not the scientific re-assessment but the standard--and put in a compliance

schedule from that, using the legal mechanism you're talking about, you are still talking about five-plus years.

STICKNEY: It seems that we are concentrating on the paper mills, where you cite this article in the Wall Street Journal, which indicates that the EPA should be focusing dioxin reduction efforts on incinerators, smelters, and oil refineries. Obviously, if people are being exposed to incinerators, such as in Portland and Biddeford and the Bangor area, why aren't more people being exposed to these air-borne dioxins than are being exposed to the problem in the rivers, which very few people get involved in? More people certainly are smelling these fumes on a continuous basis. If this article that you cite is going to have merit in this hearing, certainly that should be being discussed as well as just limiting it to the paper mills.

KREISMAN: There are at least three responses I'd like to provide. First, the Natural Resources Council--and anyone else who knows anything about this issue--has never maintained that the paper industry is the only source of dioxin and has never maintained that this hearing should be mutually exclusive of anything else. We have participated in incinerator licensing issues, for example. Second, when you talk about routes of exposure to dioxin--and I am not the expert here; certainly Dr. Silbergeld would be a person to ask that--you are talking about a molecule which then bioaccumulates fifteen thousand times in fish. You are talking about a much different exposure regime, it is my understanding, than a molecule that is coming out of an incinerator. Third, I am not sure that I agree with your presumption as to no one being exposed to this in the

rivers. The paper industry has often made the argument, using a graph that shows how little the paper industry actually discharges compared to other sources that you've cited. In terms of the absolute amounts of discharge, I am not sure anyone disagrees with that. I think where the condition comes is saying, sure, that's how much you're discharging, but what is the route of exposure? The route of exposure is in fish, which are biomagnifying that fifteen thousand times. So you are comparing apples-and-oranges. That's why EPA has so much concern about this.

STICKNEY: I didn't make the statement that no one was exposed to the rivers or the fish. One thing I did ask yesterday, which I didn't get answered, is: what chemical neutralizes dioxin, so that it becomes innocuous to the system? The state toxicologist could not answer that yesterday and I hope that maybe one of your witnesses will.

KREISMAN: I think they certainly can. If you hear any answer other than, nothing neutralizes it, it is a persistent bioaccumulative compound which constantly loads in our environment and the only thing that is happening to it is that it may get covered by subsequent layers of sediment, only to be uncovered when the Kennebec River is flowing at 200,000 cfs in April of 1987--I think that's the response you are going to hear. Dioxin has a half-life that the state toxicologist testified is around seven and a half years, I believe. But I have never heard any theory of neutralization. This is a very persistent, very bioaccumulative compound. We see that again and again in what is in the Great Lakes, etc.

STICKNEY: Just a personal observation. I can't believe that anything that can be created cannot be destroyed. If putting paper together with chlorine creates this dioxin, obviously something else can reverse it. That would be my assumption.

KREISMAN: I am so far out of my field that I am not even treading water at this point; I am drowning.

LIVESAY: When is it that you are anticipating these federal standards to be adopted?

KREISMAN: What I am anticipating is what I read. What I read is that EPA is on some schedule. Maybe it will change with the new Administration. That's a new factor. But they are talking about a human health re-assessment being done around September of 1993. They are talking about a wildlife and aquatic life re-assessment, which is showing some pretty interesting things, going to be done about a year later. At some point in that time frame, EPA has been quoted as saying it is going to take several years to develop a water quality standard.

LIVESAY: Does that suggest 1995 or '96 or '97 or something beyond that?

KREISMAN: No, that's not my understanding. But my understanding is that, even if it is 1994, then you are talking a three-year compliance schedule and implementing it, you are into 1998. And that is assuming, if it is a restrictive standard, that the paper industry will not litigate against

it--which would be totally contrary to their behavior all across the country and with EPA.

LIVESAY: What did the paper industry tell us yesterday in terms of their expectations, or did they even say?

KREISMAN: I am not sure I want to represent what they said. I think they've represented in the press... I remember Floyd Rutherford, in an article, saying, I believe, he thinks it is going to be at least another year and a half. But I don't think they were telling you there was a juggernaut that was going to solve this problem in the next four or five months. Their witnesses said that the EPA reassessment was completely open-ended, the findings weren't at all clear, there wasn't a consensus, that it was going to have to go back to the drawing board, etc.

EDWARDS: There is a state law, Chris, that I want to bring to your attention, which I think further complicates your ability to make this a two-year interim license. It is a new law that was enacted, I think, some time in 1991. It is 38 M.R.S.A. 344(1)(a). It says: "An application for a permit, license, or approval is processed under the substantive rules in effect on the date the application or request for approval is determined to be complete for processing." I think there is a potential problem if in this rule you say at a certain date a new standard goes into effect. Possibly someone could challenge that by saying the license is governed by the rules and the law in effect at the time that it was accepted for processing.

LIVESAY: It seems to me that is something that might be worth exploring.

EDWARDS: I just bring it to your attention because I think it is an issue that is going to come up. In fact, I've had correspondence on it and I'd be happy to share that with you.

Peter Washburn for NRCM

I am Peter Washburn, staff scientist at the Natural Resources Council. In addition to what Ron told you about me, I was part of a group made of IF&W, DEP, DHS, and the paper industry who designed and scheduled the 1992 dioxin monitoring program, the data from which I'll show you today.

As Ron noted, one of the first questions we had when we looked into this issue--and I'll reiterate what he said, that we thought very hard about this--was, what do the levels in fish look like? These data we have just received over the last week or two, so I'll note that the data at this point are preliminary. However, since they were shown to you yesterday rather quickly, I thought I'd use them as well. The data I am going to present is based on game fish. We have data from bass and trout. I didn't include data from rainbow smelt because they are not a resident species--they are anadromous--but we can talk about that if you have some questions about why I didn't use those data.

At Tab 5, you will find "1992 Monitoring Data--Dioxin in Maine Gamefish." I'll just point out that the rivers are down the rows, also locations where sampling was conducted. There are some averages that I can explain. The last three

columns on the right are levels of dioxin and a total level of dioxin. I know you've seen a lot of confusing tables over the last couple of days and I am sorry to add to that. Let me explain the three righthand columns. The one labeled "TCDD" is the dioxin we were talking about yesterday. In fact, dioxin is a group of compounds--compounds that look similar and act in a similar way in both humans and wildlife. The difference among this group of compounds is their relative toxicity. For example, the toxicity of TCDF is about one-tenth that of TCDD. So when you are looking at the total effect of dioxin, such as contaminating fish--the total load someone would be getting into their body--you multiply, such as in the first example for Lincoln, 2.77 x 0.1. Then you add that to 1.05 and you get 1.33. That's called a TEQ. I understand that's how you regulated dioxin in the sludge rules--to group the other dioxins as well as TCDD as part of a TEQ.

I should point out that when we developed this program the group discussed how we were going to handle the data. We discussed that TEQs were going to be calculated, and when the state toxicologist develops fish advisories it is based on a TEQ, the total amount of dioxin.

Again using the example of Lincoln, I have given two averages. There is a TEQ average and an average. The TEQ average includes dioxins other than TCDF and other than TCDD, which we didn't monitor this year but we monitored last year. The reason we didn't monitor them this year was because it was an extra $500 per sample. So it was agreed by the group prior to the sampling program that we would estimate what those other dioxins contributed for a given river.

Now we'll talk about the data. You'll notice that I've calculated for each river--Penobscot, Kennebec, Androscoggin, and Presumpscot--river averages. With the Penobscot, it is 0.81 ppt; for the Kennebec, it is 0.88 ppt; on the Androscoggin, it is 1.45. I should point out that these are fillet concentrations of dioxin--the amount in the fillet that people would eat. The total of all these data is a total TEQ of 1.08 ppt.

You'll notice that there are areas within each river, other than the Presumpscot, where there are elevated levels. This is very interesting data. You should know that the state toxicologist establishes fish consumption advisories based on his current assumptions related to the toxicity of dioxin, not what he referred to yesterday as what looks like evolving evidence that these advisories will move lower. Currently he establishes an advisory for protection against cancer effects at 1.5 ppt. He establishes a reproductive advisory at levels of 1.85 ppt. If you exceed these values, he will issue a warning. In the case of cancer, it will limit the general public's consumption of fish from the river; in the case of reproductive effects, it will be a warning to women of childbearing age. I want to point that out. A couple of times yesterday people referred to this warning for women as a warning to women who were pregnant or nursing. In fact, the state toxicologist issued the warning to women of childbearing age. That covers a lot more people and reflects what some of the witnesses will refer to later as a critical time.

Going back to the data, you'll notice that on the Penobscot at Lincoln the TEQ average is just slightly less than 1.5. You'll notice that there are two values for Augusta. The upper one is for small-mouth bass and the

lower one (2.32) is for brown trout. Those are two brown trout, which is all they have reported at this point, but the average of those two fish is greater than both the reproductive advisory level and the cancer-setting level. You'll notice that on the Androscoggin at Gulf Island Pond both of those advisory levels are exceeded. You'll notice that when you add all the data on the Androscoggin together and take the average, you are again fairly close to that advisory level.

I wondered about how advisories would be set based on these kinds of data. So I talked to Dr. Frakes and I said, how would you handle this kind of thing? I think it would be interesting to follow up this question with him, but he indicated to me that in a situation like this he would be inclined to set advisories for these areas of the rivers.

Last year you may have seen the newspaper articles and the television stories that the levels were going way down. There were calls to remove all advisories from Maine's rivers. The levels have dropped. The paper industry should be commended for that. When these data came out--and I knew that there was a certain amount of rush to have these data available for this hearing--I was expecting the values to be pretty low. But we have levels at locations in the rivers right now which, even under the existing advisory triggers, exceed them. To me, that answers the first question we had: do we have an existing problem? I just wanted to share those data with the board.

STEVENS: Any questions for Mr. Washburn?

STICKNEY: I am a little troubled because we've been led to understand that this is an accumulative thing, such as

taking pennies and stacking them one top of another; that as the dioxin moves down the river, the concentration gets greater. And yet I presume that Jay is above Livermore Falls and it drops off. Then we have Gulf Island, which is above Lisbon Falls, and it drops off. This is on the TCDF. Then also, with Gulf Island to Lisbon Falls, it drops off in that instance. So as a Board member sitting here being told that this thing keeps stacking--each paper mills adds its own and so it accumulates--yet your figures here seem to indicate that it can ebb and flow and dissipate or increase, for no apparent reason.

WASHBURN: I won't presume to be able to answer this authoritatively, but one guess is that, at least Gulf Island Pond, there is a dam there and a lot of sediment accumulating.

STICKNEY: From my personal standpoint, I would like to have someone get us this information so that we can make a more judgmental decision on this case before us, to understand whether or not it does continually accumulate until it hits the ocean where, according to Dr. Frakes yesterday, he thinks the saltwater does neutralize it. But he didn't know.

WASHBURN: I won't speak for Bob Frakes, but I think what he was suggesting was that it may sink when it gets down and end up in the sediments in estuarine areas. I think that's what he was saying yesterday. I will note that at the mouth of the Kennebec and the Penobscot, and I believe at the mouth of the Presumpscot this year, they are taking samples of clams and analyzing them for dioxins. It will be very

interesting to see that data because we have not really had that type of data in the past.

BONSEY: We had testimony yesterday that indicated that this interim rule would increase the potential amount of dioxin by 38-to-1. However, industry told us that, in spite of that, they would not in fact be putting out any more dioxin than they are today. Let's assume that's correct. What kind of effect will that have on these kinds of figures over the years, if they hold their dioxin output at the same level it is today? Will the dioxin in gamefish thus remain at this level, or will that increase?

WASHBURN: It's hard to say. We don't know whether dioxin is continuing to be reduced in the fish or whether it is leveling off. I can't answer that question. It will only be a matter of continuing to monitor the sites. There is a possibility that this is the bottom and they are not going to get a heck of a lot lower, but it is possible that it could continue to drop.

STEVENS: Was the second Augusta reading, the high level of 2.11/2.32, taken behind the Edwards impoundment? Where was that taken?

WASHBURN: I think Barry Mower could answer that.

MOWER: Just below the dam

STEVENS: Thank you

Dr. Peter deFur for NRCM

I am Dr. Peter deFur, senior scientist with the Environmental Defense Fund. EDF is a national, non-profit, environmental organization with over 200,000 members nationwide. On behalf of EDF, I appreciate the opportunity to present testimony to you today on this important topic of regulating dioxin discharges.

Let me quickly go through my academic background and tell you my qualifications to comment on dioxin water quality standards and, most specifically, on bioaccumulation and bioaccumulation factors. I have a bachelor's and master's degrees from William and Mary in Virginia, both in biology. I have several years of experience doing ecological surveys related to the power industry. I have a PhD in biology from the University of Calgary and I have completed a post-doctoral fellowship in neurophysiology, also at the University of Calgary. I have held faculty positions at George Mason University and at Southeastern Louisiana University in the departments of biology, where I have taught courses ranging from introductory zoology to graduate-level environmental physiology and ecological physiology. I have written numerous research papers, published in peer-reviewed journals, reviewed for those journals, and given seminars and lectures across the country. So I have a typical suite of academic credentials, as you can see in my résumé.

I am here to talk about water quality standards because, since coming to the Environmental Defense Fund, I have been heavily involved in state and federal activities to regulate dioxin discharges and emissions related to both ecological and human health effects. I have followed this

extensively through the re-assessment process that you've heard so much about. With regard to dioxin, my experience extends beyond commenting on state and federal regulatory activities and presenting testimony before Congress, but I have focused it on bioaccumulation.

There are three fora that I wish to refer to upon which I draw my most recent experience for bioaccumulation. Those have to do with the effort by EPA to set water quality standards for an entire region of this nation, as a region rather than simply by state or by river, and that is at the Great Lakes. The Great Lakes initiative was mandated by Congress and it required that the states get together and set water quality standards for all of their chemicals, not just dioxin but a whole suite of chemicals including metals and other persistent bioaccumulative compounds. This proposal has gone to the Science Advisory Board of EPA. Now the Science Advisory Board is a group of external scientists who are recognized experts in their field. I am a member of that ad hoc Science Advisory Board review committee for the Great Lakes initiative.

I was also invited to participate in EPA's effort to, for the first time, set water quality standards to protect wildlife. Until this time, EPA has set all of their water quality standards to protect human health. We have a lot of information about human health and, of course, this is an important end-point. But EPA is also mandated to protect other species and the ecosystem at large. They are now beginning to develop the methods, the guidelines, on how they will do that. I was an invited participant at that workshop in which those are beginning to be developed, in which we discussed models, formulas, equations, data, what do we have, what do we need, and where we're going.

Finally, EPA joined with the National Institute for Environmental Health and Safety, the Chemical Manufacturers Association, the American Paper Institute, the American Petroleum Institute, and others in sponsoring a very important working conference this summer on bioaccumulation. I'll explain a bit about bioaccumulation and why there is an entire working conference of academic specialists and the only invited nonprofit representative there. So I think you are beginning to understand, from some of your questions, how complex the whole topic of bioaccumulation is and why we would have an international conference on that.

In these fora, I have developed expertise on bioaccumulation. What I am going to tell you is a little different and, I hope, a bit refreshing because I am not going to come up here and bash the bioaccumulation number of 14,300 that is in the proposed regulation. I am going to tell you that's not really such a bad number, so that's the good news. Isn't it nice to have an environmentalist come up here and support something for a change? Let me explain. I don't believe it can go any lower. In fact, my personal and professional opinion, based on my experience in conducting ecological surveys and physiological experiments myself and reviewing and going through all these proceedings--I think it is going to get higher as we go down the road. I think when we come back in five years--if not in this forum, we will be doing something like this in another forum--we'll see data to support a higher bioaccumulation factor than 14,300, or about 15,000, or even a little more than that. We'll see one substantially larger.

I am going to explain to you why I believe that. I am also going to tell you that current developments show that those estimates for how dioxin and its related chemicals

that Peter Washburn just talked about--the congeners, the other chemicals that act in the same way--why we expect to see that and some of what goes on in the ecosystem, particularly the aquatic ecosystem, when dioxin or one of its congeners is released.

First of all, we started out a number of years ago using a simple bioconcentration factor. EPA regulated and determined water quality standards based on the simplistic experiments we started out with, which was to take a fish, put it in an aquarium, add some chemical--in this case, dioxin--and determine how much, at the end of the experiment, is in the fish and how much is in the water, and we get a simple ratio. That is where it began. Over time, EPA scientists, independent scientists, contract scientists all engaged in two efforts simultaneously: (1) to improve that. If we take a simple experiment, we should be able to improve it--improve the measurements, the measurement techniques, the way we conduct the experiment--in order to understand more about it and to get better numbers, because EPA is continually doing that. (2) Understand what really goes on in the real world. The real world is not an aquarium tank. The questions that we've had this morning so far and yesterday reveal that. You understand that the concentrations don't always follow the expected pattern. What really happens out there? So these are two lines of investigation that have been going on at the same time.

Now in the one case of improving measurements, EPA has seen, and independent scientists have seen, increasing numbers of bioaccumulation. I'll submit this in written testimony later, but I have here a table of bioaccumulation numbers. These are data that were shown yesterday. If you look at those, the earliest one, Branson's in 1985 has a

steady-state bioconcentration factor, which is the simplest form (I am not going to evaluate that; I am going to show you the trend over time), of 81,300. Then, as we progress, the 1986 one is 113,000. As time has progressed, we have numbers which are increasing. The reason they are increasing is twofold. because we understand more about what is going on in the experiment and can design the experiment better to make more precise measurement, and because the instrumentation to make the measurements has improved over time. So that in 1992 the proposal to the Great Lakes initiative included numbers in the millions for a simple bioconcentration factor, because we know more about what to measure and how to measure it.

These numbers are obtained from a number of EPA scientific studies that are already in your packet from yesterday. They are already cited and quoted by Mr. Sherman. Based on a three percent lipid, which is the Maine state basis for determining bioconcentration factors as they will be used in your water quality standard development, you'll see that the 1985 number is 2,439; and we progress down to 129,000, which is the translation from the steady-state bioconcentration factor into what is actually used in the formula. That is to adjust for the fat content of the fish and for the fact that you are regulating on fillets instead of the whole fish.

So, as you can see, in time we are progressing to larger and larger numbers. That is a bit alarming. Scientists don't expect to see these huge changes. They don't expect to see a quantum leap, an order to magnitude shift overnight. Experiments generally tend to agree with one another. So when they don't, we seek answers. The experiments revealed that there was an important difference

between how you measure things in one experiment and another, as you heard yesterday. How does that apply to the real-world situation? This is the next question that was asked: what happens in the real world?

In the real world, we know that dioxin is discharged from the end of a pipe as a complex effluent and there are a number of different chemicals. They behave a little differently. One of them has an equivalence of 1.0, another one less, another one less. They do different things. But you've also heard that some of that dioxin will stay in the water, some of it will adhere to particles and float around in the water, but a great deal of it will adhere to particles and be contained in the sediment. So as we began to understand more about how it behaves technically, we began to understand more about how it behaves biologically. Hence the need to be able to predict exactly what happens as it flows out of a pipe and down a river in Maine. How do we predict that? Do we predict that on the basis of river flow? on the nature of the bottom of the river? Is it simply the nature of what animals are living there? What are the ecological measurements that must be made? How do we know that?

We cannot make all of those measurements for every case. We have been spending ten to fifteen years trying to get to this point, where we can predict it fairly accurately because of accurate field and simultaneous laboratory measurements in Lake Ontario. We have spent a lot of money. Rather than do that, let's take the information we have, use a computer model, and see if we can estimate on that basis. That's what the bioaccumulation models are doing. So that is the other effort. If everything works, when we go out and measure it in the field and we predict it with a

computer model, they should agree. They do. That's the good news. The good news is, for data where we have experiments, computer models, and field collected data--and those are in the Great Lakes and, so far as I know, that's the only case to try and model the ecosystem.

This is what they are trying to model. This is a simplified one developed by Manhattan College. They are trying to understand how many boxes you put up there. What is the rate at which it moves from one box to the next? It takes years, not only to design that system but to make all the measurements of how many fish, which species--are there crayfish, are there clams, are there insects--which birds feed on that system, how is that system designed? Those are the efforts that are going on right now.

Everything we know, everything that scientists have learned, indicates three things about dioxin and its related chemicals. (1) It is widespread in the terrestrial and aquatic environments. (2) It is persistent. It is persistent beyond what you've heard so far. Sediments contaminated with dioxin in Newark Bay have not had measurable changes in concentration in thirty years. Dioxin discharged into Newark Bay thirty years ago remains in the bottom of that bay and contaminates seafood today. It causes abnormalities in crabs when they shed. So the dioxin discharged today will be around. (3) It moves into the living system, into this ecosystem. Those are the three important things to remember. And its movement and concentration are high.

These are developments that have occurred over a number of years. These are things that have come out of the three proceedings that I've talked about, developing an excellent data set in the Great Lakes area, consensus reached

internationally by scientists in these proceedings, and data that are being used to protect not only humans but wildlife and the aquatic ecosystem. The Scientific Advisory Board ad hoc committee reviewing the Great Lakes data was shocked to find out that the water quality standard to protect wildlife is lower than the 0.013. The Great Lakes Initiative is developing a number to protect certain birds and mink that have been locally removed from the Great Lakes because they have been feeding on contaminated fish. In order to protect those species--because some of them are more sensitive than humans--we will have to have an even lower water quality standard than 0.013. That was surprising to the scientists because we had not anticipated that and we had not seen that. The data can be confirmed through different measurements. When scientists see two people measure something in very different ways and they agree, after we check it every way from Sunday--because nobody ever agrees in scientific experiments; the point is to disagree--then we found out that, yes, there is good reason to believe that. So ecologically we have a persistent problem.

STEVENS: Any questions?

STICKNEY: Where is Newark Bay? Is that in the Great Lakes?

deFUR: That is on the coast of New Jersey, off Newark, NJ.

STICKNEY: You say that the dioxin that was deposited there thirty years ago is still present today. So what you are saying is that salt water does not neutralize it?

deFUR: Dioxin isn't neutralized. The breakdown of dioxin is either slow or perhaps nonexistent. So there isn't any neutralization in the way that we think of for an acid or a base or many other chemicals. It is a very stable compound. In order for it to be broken down, the chlorine has to be removed by some biological process. Then some of the very stable chemical bonds have to be broken. We don't understand exactly why it has these biological properties. That's a question for evolutionary biologists. It is also found in other coastal areas where sediments have been dredged and deposited off the coast. Over historical time, we have been dredging our harbors to keep them open. And as we are doing that, we monitor those dredge sites. We have found a number of them are contaminated not only with some of the things you'd expect but also dioxins and PCBs.

STEVENS: Most of our heightened awareness of dioxin came about in the Vietnam area with Agent Orange and its tremendous health implications, but this compound has been around, from what you say, for a lot longer time than we have been producing it, to our sorrow. Has anything been done deep in the sediments to indicate that this compound has been there for a great many years?

deFUR: Yes, and it's both what you'd expect and what you might fear. It isn't in the geologic record. We find that its concentration is highest at the time when our industrial processes that we know are related to dioxin production--such as incinerators and pulp and paper mills and some of our smelters and leaded gasoline production--all of that industrialization for a number of decades are when it started to rise and it peaked a few decades ago. It is on

the decline. So that's good news. The bad news is that it has been leveling off for about five to seven years, maybe a decade. So our initial efforts to make improvements--not unlike what you have heard about the improvements for this one industry--the initial efforts yield substantial success. The next increment of improvement is much more difficult.

STEVENS: In the Great Lakes, with your numbers up in the millions, that is in dioxin or other compounds?

deFUR: All of them. The numbers there for bioaccumulation alone are for just the one chemical, but when you go to measure how much is in the fish, they do both the one chemical (TCDD) and all of its congeners, so they'll do the sum. They also determine how much of the toxicity is due to this compound, that compound, and the other. In some cases, it's a PCB that's a bigger problem.

BONSEY: I'll ask the same question I asked the previous speaker. If industry does not increase its level of dioxin --and they've said they won't--does the concentration of dioxin levels in fish remain the same, or do they increase?

deFUR: I think we would expect it to stay about the same. Neither an increase or a decrease can be ruled out. Both have been seen. The increase is a shocker. Why should concentrations in fish increase? That is inexplicable and it is based on some limited field data where they have shown dioxin concentration is falling in the water but not in the fish. The reason is that dioxin is toxic at high levels, but when it drops to a lower level its toxicity is reduced such that the fish are more viable. They survive better,

which means that you have a more healthy fish which is more biologically active and better able to take up and concentrate it. Does that make sense?

STICKNEY: I would like to follow up on that inasmuch as the fish that are being caught and tested for dioxin are probably more mature fish. We saw yesterday the rapid decline that the paper mills have made in the amount of dioxin they are putting into the rivers and the level they have reached today. So isn't there the possibility that the young fish that are growing now, when they are going to take the bait and be caught perhaps would be the recipients of a lower level of dioxin than the older fish which have been caught and tested, which were getting that higher level of dioxin in the river? So that a future test of a mature fish may show a much lower level than these charts we received earlier?

deFUR: That is certainly within the line of expectation. Many of those gamefish are very active fish. They have a high rate of metabolism. In those fish, dioxin is removed from their bodies at a faster rate because they are more active. The sedentary fish--catfish and suckers--are not as active, they have a lower rate of metabolism, and they do not have the normal processes that would cause it to be washed out or flushed out of their systems at a higher rate. So, at some period of time, you'd expect it to drop. But then, after probably six months to a year, we wouldn't expect those fish to see any further declines until you see an additional decline in exposure or the amount being added into the fishes' ecosystem. So, yes, but...

STICKNEY: That leaves me even further confused because you say, thirty years ago the deposit in Newark Bay has remained the same, dormant, and yet you say a fish will flush out dioxin that is in its system today. If it was put in pure water tomorrow, the dioxin level will be reduced?

deFUR: That is correct. Tomorrow they won't be put in pure water, though. The issue is that the fish take up a lot from those sediments that are dormant. So as we drop the amount that is in the water, from which they get only a limited aount, the very readily available dioxin that has been freshly deposited will be flushed out. But then there will be a continual loading to the ecosystem of the amount which is in these sediments that are dormant. That's why they are trying to use computer models to predict that because, as you would expect, that process has to be different for a lake and for a river, and for a mountain river and a coastal river. That is correct and that's what we expect. However, many of these predictions and expectations have not been tested because we've only been establishing the theories and asking those questions for about five years or so. As you've heard, many of those measurements are very difficult to make, particularly the dissolved concentration.

GENDRON: We had a criterion calculation yesterday with a bioaccumulation of 14,300. However, I notice that EPA in 1984 had a bioaccumulation of 5,000.

deFUR: The 5,000 is based on earlier data that I showed you from Branson. They get a total bioconcentration factor and then adjust that for what the conditions are that they

expect are closer to reality for the situation they are regulating--fillet, lower percent lipid fish, higher percent lipid fish, whatever. That's where they came up with 5,000. The 14,300 is based on the newer data and making similar adjustments both to the fillet, which you would expect to have a lower lipid content and have a different portion of the dioxin load in the fish, and also based on the three percent fish lipid concentration for Maine fish, because that's the state toxicologist's estimation of what the condition is here.

GENDRON: Does EPA accept that also?

deFUR: Yes, EPA would accept that. In fact, EPA has made comments in some of the proposed regulations and permits at the state level, saying, you know, you could put in some state-specific data. Two states that I know of--and probably three or four more--were faulted for not putting in state-specific data.

STEVENS: Any other questions? ... Thank you very much.

KREISMAN: One quick wrap-up comment on that. If you turn to Tab 6, you will note that we have run a chart based on discharge levels going up to the allowable level in the permit--without any representation that either they will or they won't. Using a bioaccumulation factor, the righthand column will show you the allowable fish concentrations that we would expect to predict. You can compare to what you saw Peter Washburn present as what is happening right now.

The only other comment I would add is, in asking Dr. deFur about what would happen to the fish, over the next

five-to-seven years I guess, there was a premise that the paper industry's discharge levels would be going down. I don't think that premise is at all in the record. This rule allows the paper industry to operate at a steady state, using their existing technology.

STICKNEY: We received a chart yesterday showing the drop from 1988 down to the present.

KREISMAN: That's right. That's based on installation of technology between 1988 and the present. What the paper industry wants in this rule is not having to do anything further until the re-assessment is done. So I just want to make it absolutely clear that I do not believe there are any representations on the record that the paper industry has demonstrated that discharge levels would continue to go down based on additional treatment, and that therefore (without going into bioaccumulation) you can expect fish levels to go down. I just wanted to make that very clear.

Moving on from what is in the fish and what is expected to be in the fish, our next witness is Dr. Barbara Knuth. As I indicated earlier, Dr. Knuth was first retained by the NRCM after ChemRisk released its study on fish consumption rates. While I will let Dr. Knuth introduce herself, I have never had the experience of trying to obtain a witness where you call knowledgeable people all across the country and they all say the same person. So that's who you are looking at in Dr. Knuth.

Dr. Barbara Knuth for NRCM

I will be addressing the Board on the topic of appropriate fish consumption rates for Maine sportfishers. What I'd like to do is briefly go through some of my background, so that you have a sense of why it is that I feel I am qualified to be before you today and can hopefully place in some context the remarks that I'll be making today.

As far as my education, I have a PhD in fisheries and wildlife science from Virginia Polytechnic Institute and State University of Blacksburg, Virginia. I am currently a professor of natural resource policy and management in the Department of Natural Resources at Cornell University. I also serve as co-leader of a nationally known group called the Human Dimensions Research Unit in the Department of Natural Resources, College of Agriculture, at Cornell University. The Human Dimensions Research Unit has had an ongoing research contract for fifteen years with the Department of Environmental Conservation in New York to conduct research on fishing and wildlife recreation and fishing and wildlife resources. I teach and conduct research on the human and policy dimensions of fisheries and wildlife management. One of my specific areas of expertise is in human perceptions, attitudes, and behavior associated with sport fisheries affected by chemical contaminants. In the past five years, I have been awarded grants and contracts for twelve separate studies of fisherman or angler behavior associated with health advisories.

I have worked with the U.S. Environmental Protection Agency in several capacities. I currently serve as principal investigator for a major study of angler response to health advisories in the Ohio River Valley. I have been

an advisor and continue to advise EPA regarding their research agenda on what people know about health advisories and how people respond to health advisories, how human behavior changes in response to contaminants and health advisories, and how to properly conduct research to measure human behavior related to fishing and eating fish. At the invitation of EPA, I was a member of an expert panel for a state personnel training session, where the state personnel were from water quality and health agencies, and the purpose of the training session was to train these people on appropriate methods to assess subsistence and recreational fish consumption.

I have worked with other state and federal agencies and with industry groups. These are listed in my résumé, but I'd just like to share some examples with you. This year I worked with the Electric Power Research Institute, an industry group, advising them at their invitation on the topic of policy implications associated with the use of health advisories for contaminated fisheries. I have worked with the Great Lakes Council of Governors' task force on fish consumption advisories. Most recently, two weeks ago I was in Dearborn, Michigan, to report the results of my research program to state health, water quality, and fishery management staff and EPA representatives. I have worked and advised with the International Joint Commission, which is an international body composed of representatives from U.S. and Canadian federal governments, that advises both federal governments on natural resource and other policy. I have also worked with Cooperative and Sea Grant Extension on the topics of fishery resources, tourism, and local and regional economic growth and development.

My testimony today will address three major points. First, I'll address what is known about fish consumption rates in Maine. I'll specifically address the paper industry study of fish consumption that you heard reported yesterday. Although that study is flawed--and I'll explain the reasons for my conclusion that that study is flawed--and should not be used for regulatory purposes, I will review what that study says about fish consumption in Maine. The flaws in the study, however, are ones that I will present to you that lead to under-representation of fish consumption in Maine.

One of the concerns raised by Dr. Boyle yesterday and others was that the paper industry study did not assess fish consumption rates specifically for river anglers downstream of kraft mills. One such specific study exists. The paper industry, as far as I understand, is aware of that study and I am a bit surprised that no one mentioned it yesterday. That study was conducted by the Penobscot Indian Nation to assess use of the Penobscot River, including fish consumption. So if you are looking for a specific study of a population that is downstream of a kraft mill, that may be the target that you are looking for. I'll refer to that study later, but I won't go into the details because I understand you will be hearing from representatives of the Penobscot Nation who can speak to it in more detail.

The second major point of my testimony will address how fish consumption in Maine may change if pollutants were not a problem. Third, I'll address what fish consumption rates should be considered as the basis for policy in Maine and why these consumption rates are appropriate.

I am testifying today, as I mentioned, to recommend to the Board an appropriate fish consumption rate based on my

expertise and my own research and experience. The fish consumption rate, as you've already heard, is a critical underlying assumption in estimates of human exposure to dioxin and, therefore, estimates of human health risks from dioxin. Fish consumption rates are important not only for estimating exposure where cancer is concerned but also for reproductive effects determination, which you will hear about later this morning.

I'll identify and critique the assumptions underlying the paper industry fish consumption study and raise a question for the Board whether the assumptions that that study made were appropriate assumptions to be used in setting policy. One of the reasons related to my agreeing to be retained on this issue is an interest I have in seeing scientific information used appropriately in policy decisions. As I teach in my classes, too often we see value judgments masquerading as objective science. It is important to identify the assumptions on which all scientific methods and conclusions are based and ask if those assumptions are the correct ones to be used as policy is being debated. Science as well as policy is based on choices. Researchers make choices all the time that affect the data gathered and therefore the kinds of conclusions that will be, or even can be, reached.

For example, were the methods used in the paper industry study and the assumptions on which the conclusions were based sufficiently protective of the part of the Maine population that should be protected? The question of what people should be protected by pollutant standards, and to what degree they should be protected, is a value judgment to be made by you, the Board, as policy makers. But even the choice of methods used in the study--the choice

specifically of which questions to ask and how to ask them--reflects value judgments that affect the type and quality of data generated. I'll be addressing those and other issues with you this morning.

To address fish consumption rates in Maine, I'd like to share some overall conclusions and then lead into some specifics. As a policy decision, which I've referred to already, it is important to choose fish consumption rates that will sufficiently protect the human population. As you heard from Commissioner Marriott and others, fish consumption is a critical value in the water quality criterion calculation. Yesterday Board member Roy asked for information about the percents or numbers of people who may be affected by these decisions. You heard the term "percentile" used. I'd like to explain that term. Percentiles indicate what percent of the population would be protected by using a specific fish consumption rate. The use of percentiles is based on selecting a segment of the fish-eating population that will be protected by water quality regulations. The percent of the population above a given percentile will <u>not</u> be protected by water quality regulations.

For example, if we look at the paper industry study, we can estimate that there are at least 92,961 people who fished Maine rivers in 1989 and ate fish from the rivers. We do not know legitimately how many of these anglers actually fished downstream of a kraft mill. However, these are the only data that are available from that study--data on river anglers who ate river fish. Choosing the 90th percentile of that group for fish consumption would mean that ten percent above that 90th percentile, or 9296 people, eat more fish than that consumption rate. Therefore, a

water quality standard based on the 90th percentile of fish consumption would not protect the health of 9296 people, because these people would be eating more than the amount of fish that is provided for in that regulation. Fish consumption by the most frequent fish consumers can be considered the most sensitive use of Maine's waters, which you heard referred to in the legislation this morning.

Using percentiles that are based on current fish consumption does not include how much more fish people will eat in the future, once health advisories are lifted. The reason it is important to consider what people would eat is because evidence exists nationwide that people do not eat as much fish from waters with health advisories on them as they would eat if the health advisories were lifted. Once health advisories are lifted in Maine, fish consumption from those waters will likely increase. Therefore, current fish consumption rates are less than future fish consumption rates in the absence of advisories in the future.

The fish consumption study commissioned by the paper industry has serious flaws, specifically in the way questions were asked in the questionnaire and in the way the data were analyzed and interpreted. The basic operation of the sampling and mailing procedures that Dr. Boyle explained in some detail and referred to as the Domen [?] method are in fact state-of-the-art procedures. I don't dispute that. I applaud them for using that kind of detail in their mailing and contacts with people. The flaws that are of concern, however, relate to the actual questions that were asked of people and how those responses were interpreted.

As I pointed out earlier, regulatory fish consumption rates involve choice. I want to take issue, however, with the claim that was made yesterday that hard science is not

involved in this process. Hard science can be and should be used to identify the range of fish consumption rates that exist within any given human population--in this case, Maine river anglers. Describing the range of probable fish consumption rates is a matter of science. The reasons for choosing a particular fish consumption rate within that range is not a matter of science; it's a policy choice. It is a policy decision for the Board to determine if the State of Maine should protect frequent anglers or infrequent anglers and fish consumers. The serious flaws of the industry study lead to under-estimation of actual fish consumption in Maine. I'll discuss those flaws briefly in a few minutes.

My conclusions about the flawed nature of the industry fish consumption study were also arrived at, as you heard referred to by Ron Kreisman this morning, by two other experts who were working independently of me and independently of each other. The two independent experts were invited by the Maine Department of Environmental Protection. One of these was Dr. Patrick West, a professor of natural resource and environmental sociology at the University of Michigan. Dr. West had been supported by the Michigan State Toxics Substance Control Commission to conduct a statewide assessment of fish consumption rates in Michigan. The other independent reviewer invited by DEP was Dr. David Cleverly, a staff environmental scientists from EPA in Washington, D.C. In his report to the DEP, West concluded that the methods in the industry study "tend to bias the study toward low g/person/d estimates for standard setting," producing results that are "so far below almost all other credible fish consumption studies that the study should not be used as a basis for setting standards in

Maine." Then from Dr. Cleverly, the major discrepancies between the fish consumption rates reported and the rates reported in any other reasonably-well-designed fish consumption survey indicate the paper industry study greatly underestimates fish consumption.

Just as an aside, earlier this week I spoke with William Farland of EPA, whose name you've heard mentioned a number of times--yesterday by Mr. Boxer, indicating Mr. Farland was the EPA leader on the dioxin re-assessment effort. I asked Mr. Farland about the status of the fish consumption rate recommendation associated with the re-assessment. He referred me to the person he termed the EPA expert on this issue, Dr. David Cleverly. Dr. Cleverly stated that 6.5 g/day is totally inadequate and he anticipates that 30-120 g/day will be the suggested range of values for recreational fisheries, and 300 g/day will be suggested for subsistence fisheries.

So now turning to Maine, what do the paper industry data show--recognizing that those data are most likely underestimates of true fish consumption? To address Board member Roy's question yesterday, I'll refer both to the number of people affected by potential decisions and the percentiles. The paper industry study in the ChemRisk report recommended using 1g/person/day, or about one and a half meals of fish each year. This means people in Maine could not safely eat two fish meals a year from these rivers and expect to be protected by the water quality standard. Yesterday, Dr. Boyle translated the 1g/person/day value into 2.7 pounds of fish. What he was referring to was 2.7 pounds of whole fish, right out of the river. One gram per day actually means 12 ounces of fish can be consumed within a year's time. People eating any more than this amount would

not be protected by the standard based on this rate. This value of 1g/day recommended by the industry is the median, or the 50th percentile, and therefore would not protect the other fifty percent, or 46,480 anglers who currently fish Maine rivers and eat fish from those rivers. Yet this is what the industry proposed in the ChemRisk report.

If the Board decides it is acceptable to leave ten percent of current river anglers who consume fish unprotected, that would leave unprotected at least 9300 of the most avid anglers who currently fish Maine rivers. Then the Board would choose 6.1 g/day, or about ten meals of fish each year--again based on the paper industry study. If the Board decides that it is acceptable to leave five percent of current river anglers who eat fish unprotected, the Board would choose the 95th percentile. This, based on the paper industry data, would amount to at least 4650 anglers who would not be protected at the 95th percentile, which is 12g/person/day, or about one and a half fish meals each month. If the Board looks at Native American population, and decides that it is acceptable to leave the top five percent of Native American anglers who currently fish Maine rivers and currently eat fish, based on the paper industry study, the Board would choose the 95th percentile of fish consumption by Native Americans, as reported in the paper industry study. That 95th percentile for Native Americans would be 22g/person/day, or about three meals of fish each month.

What do the data from the Penobscot tribe show specifically about Native American fish consumption from the Penobscot River? Again, my remarks are based on seeing a report of that study and I think you'll hear more details about that later today. If the Board decides, based on the

Penobscot River data from the Penobscot Nation, to leave the top ten percent of the Penobscot tribe unprotected, the Board would choose the 90th percentile for fish consumption for that specific site. That 90th percentile (again remembering ten percent would still be unprotected) for the Penobscot Nation is 32.4/g/day. You've seen that figure before. That's one meal of fish each week.

Note that the fish consumption estimated by the Penobscot tribe is higher than the fish consumption by Native Americans estimated in the paper industry study. The 95th percentile for the Penobscot tribe is about three meals per week, compared with three meals per month based on the paper industry data for Native Americans, which is further evidence that the paper industry underestimated fish consumption.

How did the estimates for fish consumption in Maine compare to other locations? I chose two states to report to you because states that have fairly good estimates of fish consumption. In the Michigan study conducted by West, the average total fish consumption was 19.2/g/person, about two and a half meals per month. The 95th percentile was about 70g/person/day--slightly more than two meals a week. In Wisconsin, the average sport fish consumption was 12g/person/day--about one and a half meals a month. The 95th percentile in Wisconsin for sport fish consumption was 37.3g/day--slightly more than one meal per week.

Data from these other studies support the notion that the industry study underestimated fish consumption in Maine. But Dr. Boyle yesterday noted that we should use objective criteria to compare data from other states to Maine. So what I propose is to offer you some objective data as to how Michigan and Wisconsin compare to Maine. Based on data from

the U.S. Fish & Wildlife Service survey of fish and wildlife associated recreation, Maine anglers spend more days freshwater fishing than anglers in Wisconsin and almost as many days as anglers fishing in Michigan. Maine is the highest for the number of days of freshwater fishing by state residents in their own state, indicating use of local waters. More Maine anglers come from a rural area than an urban area, as in the other states. Research on rural culture shows that rural people have a greater tie to the land and so make greater use of its resources, including fishing, than do their urban counterparts. Therefore, Maine anglers appear to be at least as committed to fishing as are anglers in Michigan and Wisconsin, if not more so.

So the conclusion on this section is that, even if the Board bases its decision on a flawed study that has most likely underestimated Maine fish consumption rates, that study still shows that thousands of people catch and eat fish from Maine rivers. Choosing the 6.5g/person/day fish consumption rate will leave at least 7437 current fish-eating river anglers unprotected on the basis of their exposure to dioxin.

Some of the flaws in the paper industry study I'd like to review briefly. The paper industry study had a series of poorly worded questions in it. Dr. Boyle yesterday showed Q.24 to illustrate the list of species that were included in the questionnaire. It was interesting that he did not choose to show you Q.23, which asked about the number of fish eaten, "by you and/or a household member." The way that question was worded, it could include fish eaten by the respondent or by some or all other household members, or by the respondent and other members of the household. Credible studies never use an "and/or" question such as this,

especially when trying to assess the behavior of individuals as the basis for setting water quality standards. You heard Dr. Boyle say that criticisms that I had offered previously in written comment to DEP were legitimate. I criticized the paper industry assumptions in their calculations that every household member who ate Maine fish at least once would eat fish at every meal. He went through a sensitivity analysis to illustrate the effects of not making the assumption and assuming that all fish that were reported in the study were eaten either by the respondent or by just a portion of the household.

My point in bringing that up again, even though Dr. Boyle addressed it, is to indicate that there were several flaws that, by themselves, seem to have a minor influence towards underestimating fish consumption but, taken together, compound the dangers of underestimation of fish consumption. Other studies ask specifically about fish consumption per person, rather than assuming that anyone who ate fish once would eat fish at every meal. So there are other methods that could have been used.

Yearlong recall of highly detailed information, used in this study, is inaccurate. Asking people to remember the amounts of fish they ate over the preceding twelve months is not usually done. Other fish consumption studies use daily diaries or shorter recall periods, such as seven days or one to four months. The Westat U.S. Fish & Wildlife Service study that Dr. Boyle referred to indicated that "frequent fishing trips that take less than a day are more difficult to remember than multi-day trips." If Maine anglers are likely to fish locally, or if frequent fish consumers are likely to fish for short periods of time but often, these fishing trips and the fish caught on them, and therefore the

fish eaten, are likely to be remembered less well than long, eventful, or unique fishing trips.

Yearlong recall problems are compounded by asking anglers, as the paper industry study did, to remember what all members of their household ate over the preceding year, at or away from home. How could they possibly know this? Could you answer for your family what every individual in your family ate as far as fish consumption over the past year? where they ate it? and the size of those fish eaten? Questionable.

The design of the study does not represent all potential fish consumers in Maine. For example, unlicensed anglers who may be subsistence anglers, or future fish consumers; for example, people who no longer fish or eat fish due to pollution but would if the water was cleaner. These people may, or may not, currently buy fishing licenses. Only those who had a fishing license in 1989 were among the people for whom fish consumption was assessed. So in the absence of health advisories in the future, how much fish may people eat?

First of all, why is it important to consider how much fish consumption would increase when health advisories are removed? If removing a health advisory has the effect of changing people's opinions about the quality of fish and of the fishing experience, people who stopped fishing or eating fish from those waters may start eating fish again, or they may increase their current consumption that they are now limiting because of concerns about pollution. Imagine that a pollutant standard is set based on a hypothetical fish consumption rate of 6.5g/day, which was chosen to reflect current consumption in some area. Imagine further that the health advisory on a body of water is removed. What if

removing the health advisory has the effect of changing people's opinions about the quality of the fish and of the fishing experience, so not only do more people begin fishing again but those who are already fishing begin catching and eating more fish--more than the original 6.5g/day rate on which water quality standards are based? Policy makers should anticipate what these new fish consumption rates may be. If fish consumption rates increase beyond what is used as the base assumption from which to calculate health risks, health risks will be underestimated.

How do people respond to pollution and health advisories in other states? Studies in Kentucky, West Virginia, Michigan, New York, Wisconsin, Minnesota, Ohio, Pennsylvania, Illinois, and Indiana demonstrate that anglers--anywhere from about 16 to 67 percent of them--change their participation in fishing activities, including reducing the frequency of eating fish, or ceasing to eat sport fish altogether, as a result of health advisories. All of these states, except New York, have health advisories on only some of their waters, similar to Maine. Dr. Boyle was mistaken when he claimed yesterday that all Great Lakes states have a blanket health advisory on all of their state waters. That is not true.

How do people respond to pollution and health advisories in Maine? Within the Penobscot tribe, according to the Penobscot data, sixteen percent do not use the Penobscot River at all due to concerns about pollution. Sixty-seven percent have concerns about eating fish from the Penobscot River, mostly because of pollution. The paper industry questionnaire, the ChemRisk study, included several questions that could help address this issue specifically for Maine: how much are people not eating, or what kind of

avoidance mechanism do they have, because of health advisories? Those questions were in the questionnaire. The data should be available. But those data were not reported in the July 1992 ChemRisk report on fish consumption.

I was surprised yesterday that Dr. Boyle reported that Commissioner Vail of Inland Fisheries & Wildlife does not believe that consumption in Maine waters is being suppressed, because data from the industry study indicate that in fact it is being suppressed. Although the data were not included in their written report, ChemRisk personnel hired by the paper industry said to the Department of Inland Fisheries & Wildlife in a letter that "73% of the respondents [to the questionnaire] said the health advisories affected whether they kept the fish caught from the affected waterbody, with most reporting that they kept no fish from those waters." On a different question, sixty percent (60%) of the respondents said that "they eat no fish from those waters" with health advisories. So, clearly, fish consumption suppression appears to be occurring in Maine.

Now I'd like to go to my four conclusions. What should be the recommended fish consumption rates for use in Maine policy? The Board will see the fish consumption numbers I am going to recommend or review in my conclusions later today in other health risk calculations. First of all, let's address the 6.5g/person/day consumption rate. That rate is about ten meals of fish per year. It is based on the consumption of commercially and recreationally caught fish from fresh and estuarine waters. It represents the average per capita consumption rate for the entire U.S. population, including people who do not fish and people who do not eat fish. Therefore, that 6.5g value underestimates

the per person consumption of actual fish consumers. Furthermore, that value is based on data from the late 1960s and early 1970s and does not reflect trends among most people towards increasing fish consumption. Therefore, the 6.5g/day fish consumption rate is inadequate for recreational fisheries. This rate would protect less than fifty percent of Native Americans fishing the Penobscot River and is considerably below the average fish consumption rates reported in other credible fish consumption studies.

Second, if the Board seeks to protect the 95th percentile--meaning that five percent of river anglers currently eating river fish, 4648 people according to the paper industry study, will not be protected--the Board would choose 12g/person/day. The paper industry study is likely an underestimate of actual fish consumption and does not address likely future fish consumption if health advisories are lifted. This rate is at or below average fish consumption rates reported in other credible fish consumption studies.

Third, if the Board seeks to protect Native American river anglers who are currently eating fish, the Board could choose the 95th percentile from the paper industry study. According to that study, this would be 22g/person/day, about three meals per month, and would still leave five percent of current Native Americans who fish rivers and eat river fish unprotected. According to the Penobscot Nation study, 22g protects somewhere between the 75th and 90th percentile of the Nation, based on those river data. The Penobscot Nation study indicated the 95th percentile would be 97g/person/day, or three meals per week. Likely future fish consumption in the absence of health advisories is not addressed in either of these studies, the Penobscot or the paper industry.

My last point. If the Board seeks to protect a river angler who, either under current or near-future conditions, would eat one meal of fish per week, the Board should choose 32.4g/person/day fish consumption rate to protect from undue human health risks a sensitive use of Maine's river waters. Even using 32.4g/day would leave at least ten percent of the Penobscot Nation river fish consumers unprotected.

STEVENS: Questions?

ROY: I appreciate the comments you made regarding the ChemRisk study. I am happy to hear about those because I had a number of questions about that study yesterday. One of the things you said was that you had some concerns about how people could recall what they ate. My recollection is that Dr. Boyle really felt quite strongly that the inclination would be to overestimate fish consumption and not to underestimate it. I am not sure why he was thinking that--because all fishermen tend to exaggerate?--or what he used for criteria, but I wonder if you could comment on that?

KNUTH: There was a study performed by a company called Westat that was hired by U.S. Fish & Wildlife Service to assess recall bias questions. They did a thorough review of the literature on all sorts of research--not just fishing activity--that addresses recall bias. The vast majority of studies say that, the longer the recall period, what happens is an underestimation of activities. There haven't been, however, very many studies that have been done specifically on fishing activities. There have been three that I am aware of. Two of those studies say that a longer recall

period tends to overestimate fishing participation. They didn't assess fish consumption. One of those three studies said that longer recall period (12-month recall) tends to underestimate fishing participation. So based on three studies, I am not sure that we have a clear mandate as to whether it is overestimation or underestimation. Studies have shown, though, and the Westat study that Dr. Boyle referred to does include information that says that those activities that are not that glorified--for example, taking a five-day trip to go Atlantic salmon fishing would probably be a pretty eventful experience in some people's lives, so they would tend to remember that. But if you do something that is a very frequent activity, a very normal activity--going out fishing for a couple of hours after work on the Kennebec River, if you live in Augusta--those kinds of activities tend to be very difficult to remember specifically. That's where the Westat study reports that there could potentially be underestimation problems associated. So I would say that the jury is out in terms of overestimation or underestimation with clear information as to what percents that in fact may be. What it does say, though, is that twelve-month recall has recall bias associated with it. And so any other credible fish consumption study, especially those that are being used for regulatory purposes, do not use twelve-month recall. The West study, for example, that was sponsored specifically for regulatory decisionmaking, used a seven-day recall approach. Right now in New York we are testing two methods. We are testing a yearlong recall and a daily diary approach to get some handle on what the discrepancies may be in under-reporting or over-reporting. We don't have those data available right now. My point in raising that was that

there clearly are questions about the appropriateness of choosing that kind of recall period for a regulatory fish consumption study.

ROY: Thank you. My other questions relate to the Penobscot Nation study. We don't know an awful lot about the methodology of that study at this point. I understand we will, but did you review the methodology of that study as scrupulously as you did the ChemRisk study? Did you go into as much detail in reviewing that?

KNUTH: No, I did not go into it in as much detail. What I do know is that the tribe did attempt to conduct a census of their nation, meaning that all members of the tribe were sent a questionnaire. About 25 percent of the tribe responded. Now that sounds like a low response rate, but realize that this is the entire population. The paper industry data certainly didn't hear from 25 percent of the whole population of river anglers. But do realize that that was the 25 percent who chose to respond to that questionnaire. And so we have very good data, very solid evidence, for 25 percent of the Penobscot Nation on those questions. We don't necessarily know how representative that is of the other 75 percent, but that could be done by looking at the characteristics of the people who responded to the questionnaire, looking at their demographics, looking at their behavior, looking at where they live. You can make some judgments as to how representative that would be of the entire Penobscot Nation. I have not done that.

ROY: But there is no real way to translate the information that came out of that study to any sort of statewide consumption figure?

KNUTH: No, I would not make that leap because my understanding was that that was for registered tribe members. I may be incorrect on that, but that's my understanding. I think that you will hear later today some of the Native American groups who were not even asked about their fish consumption in that study may be in fact heavy subsistence users. I think the Penobscot representatives will address that and I don't want to go into that because I am not familiar with who was missed in that particular approach.

STICKNEY: Dr. Knuth, you highlighted the figure 92,961 people fishing Maine rivers in 1989 from a paper industry study. That was not identified that it was on the four rivers with paper mills. Is that the case, that these people fished only those four rivers?

KNUTH: No. What I said was that the paper industry study only provides data on river fishers in Maine. I am not saying that these were only those four rivers that have kraft mills on them.

STICKNEY: You are from New York and VPI. We learned in geography at an early age that we have five thousand rivers and streams in the State of Maine. Now there are only four rivers that have paper mills on them. So that leaves us 4996 rivers and streams that could have unpolluted fish. So I am just questioning whether or not your statistics, based on that assumption, have much validity for the rest of the

almost five thousand rivers and streams which are populated with fish. Ms. Roy is in charge of the Saco River Corridor Commission. There is no paper mill on there and I am sure there are a lot of fish in the Saco River. We have the Piscataquis River, which runs between New Hampshire and Maine. That doesn't have a paper mill. We have the Allagash. We have many other rivers. I am sure there is great validity in the things you have said, if it involves those four rivers, but with almost five thousand others to use, I think this thing is not in as great depth as it should be.

Also, how many days or weeks do you use in your statements on consumption? Do you use it on a twelve-month basis? Because if you do, we have state laws that close the rivers. Not being a fisherman, I don't know what the last day is, but they don't open again until the first of April each year. So you obviously can't use December-January-February-March in your calculations.

KNUTH: The data that were reported by the industry study, which are the percentiles I used, were based on twelve-month fish consumption. Those grams-per-day estimates are based on fish consumed over the legal fishing season and the legal fish caught and kept in somebody's freezer that would be spread over the year. In fact, people do catch fish in those open seasons and keep that fish for later consumption. We have evidence of that not only from New York State but from many other states, that people can freeze and otherwise preserve their fish for later use. So in fact sport fish consumption, while it is only caught in a portion of the year, does occur across the year.

For regulatory purposes, the grams-per-day estimates are what are used for the calculations. The estimates, though, in the industry survey as well as other kinds of studies, take what people actually report--in this case, I believe it was under-reported--and then break that down, divide it by 365 days, and get your per-day estimate.

As far as your question about the numbers of anglers who are fishing on those four rivers of primary concern, no, I don't have those data. The paper industry doesn't have those data. That was the reason, I think, that the paper industry made reference to needing information from a specific body of water whose anglers are specifically downstream of kraft mills. That is why I would ask you to pay very careful attention to the one specific study that I do know exists. That is the study of the Penobscot Nation.

STEVENS: Bearing on the 92,000 anglers who ate fish, with the trout and salmon catch-and-release activities, are you comfortable that that paper mill study is a reasonable figure on which to base both the number of anglers and the fact that that number consumed the fish?

KNUTH: As far as I can tell from the information that was made available to me, the sampling strategy that was used in the paper industry study to sample licensed recreational fishers is adequate. In the questions that were asked on whether or not you fished a river, those questions seemed to be adequate. So, yes, I would stand by my calculation of the 92,000 as being anglers who currently fish rivers and currently eat fish out of those rivers. Those are translated specifically from the ChemRisk study. What I am not comfortable with are the fish consumption questions that

were highly detailed and I think had misleading or uncertain wording in the questions.

GENDRON: Yesterday somebody brought up the scenario of a young man--we'll say eight-nine years old--walking down the road with a catch of fish. We are talking about licensed anglers, but I think there are equally as many young boys and girls who are out there. You see them all the time walking up and down the road with a fish pole over their shoulder.

KNUTH: That was my point. Just the sampling structure of how the people were contacted to be able to answer the questionnaire was based on anglers who had a fishing license. Granted that the paper industry study was supposed to ask people what did other members of your household report, in fact, especially for twelve-month recall, would you expect anyone to know what their 8-year-old or 15-year-old or any other child caught, ate, the size of the fish, over the past year? No, I would guess not. As I indicated, all other credible fish consumption studies will ask about a shorter recall period, for individual members of the household, and try to do it in such a way that people are aware of the fact that they are supposed to be keeping track of the kinds of fish consumptions that individuals in their household are engaging in--rather than being asked at some point in time to reflect back on the last twelve months, for which you really had no reason to be remembering, what people were eating. There also are, as I understand it, not only children who may be unlicensed anglers but there may be some other groups, such as tribal members with sustenance

rights. Again I don't know the details on that, but you can ask those questions of others later.

STEVENS: Any other questions? ... Thank you very much.

Thomas F. Webster

My name is Tom Webster. I am a research associate at the Center for the Biology of Natural Systems at Queens College, City University of New York. I have a bachelor of science in interdisciplinary science-biophysics from the Massachusetts Institute of Technology. The Center where I work focuses on problems associated with toxics compounds, solid waste, and energy policy. Dioxin has been one of my research areas during nearly nine years at Queens College. I have had papers at the last eight international symposia on dioxin and related compounds as well as other scientific meetings. The topics of this research include the origins of these compounds, assessment of human exposure, estimation of the levels found in humans and its implications, and cancer risk assessment. My c.v. is at Tab 9.

In September, I served on the U.S. EPA's scientific panel reviewing the agency's current re-assessment of dioxin. I was one of the invited reviewers for the draft chapter on animal carcinogenicity. I also worked on a subcommittee discussing body burdens and reproductive and developmental effects. The testimony I'll present today reflects my conclusions based on my own research, the scientific literature, and some of the work of the EPA panel. Let me add that I am appearing today at the request of the Natural Resources of Council of Maine, which is

paying my expenses but no fee or honorarium. My written testimony is at Tab 9. Then there will be some tables I'll be referring to at Tabs 10 and 11.

Why am I talking here today? I understand that yesterday you heard nothing about the so-called background body burdens of dioxin or about so-called background doses. These are important concepts I'd like to acquaint you with. The so-called background body burden is the amount of dioxin and dioxin-like compounds which we find in the average person--not necessarily a person who is exposed to dioxin in a factory but someone like you or me. We call the background dose the dose to which the average person is exposed. Now many people are surprised to hear that the average person carries around a certain amount of dioxin in them, although I think in the scientific community this is now generally accepted.

Where does this come from? We think it comes mostly from food, particularly fat-containing foods such as meats, dairy products, fish, eggs, and poultry. Later in my testimony, I will discuss both the body burdens that I think we might expect for people in Maine, what the background dose will be for people in Maine, and what contribution might be coming from fish for fisherpeople in Maine.

Why else is it important to look at body burden? You need to know that exposure from fish in Maine rivers does not take place in a vacuum but in fact it is adding to other sources. This has important toxicological implications, as Dr. Hughes and Dr. Silbergeld will tell you later.

I'd like to start off by talking about average body burdens of dioxin, the prevalence of these compounds both in the average American and in the average resident of Maine. As we heard before, what the public commonly thinks of as

dioxin--compound 2,3,7,8-TCDD--is actually only one member of a fairly large family of chemicals. By dioxin-like compounds, I will be referring to those chemicals which are thought to operate by the same toxicological mechanism and to cause the same spectrum of effects. The total amount of dioxin-like compounds is generally expressed as an amount equivalent to TCDD. That is called dioxin equivalents, or TEQ for short. Peter referred to that earlier.

Now due to a shortage of information, the data presented by me here today are limited to the general category of dioxins and furans. Inclusion of other dioxin-like compounds--for instance, certain kinds of PCBs, which are quite abundant in the environment--would increase the average body burden above what I am telling you about today. In fact, use of the most conservative--and probably too high--equivalence factors for those PCBs would approximately triple the human levels of dioxin-like compounds. So it is worth keeping in mind that the numbers I am giving you are actually on the low side for the true levels.

The average citizen of industrialized countries, including the U.S., carries in their bodies a certain amount of dioxin and dioxin-like compounds. This is sometimes referred to as the background level. Let me just walk you through this table ["U.S. 'Background' Body Burdens Measured in Three Recent Studies"]. These were three studies that were done in the late Eighties--some of the most recent data on levels of dioxin and dioxin-like compounds in Americans. These are the actual concentrations found in body fat, or adipose tissue, or in blood. The first one, NHATS, is the National Human Adipose Tissue Survey, which is an EPA program where they take samples of body fat from victims of traumatic accidents, also elective surgery. So it is a way

of sampling body fat from around the country and looking for different kinds of chemicals, including dioxin. They found levels of 5.38 ppt of TCDD in that body fat. Now when you look at the total TEQs of dioxins and furans, it's around 27.9. So that, as best we know, is the average level of those compounds in the body fat of Americans in 1987. As a comparison, we have a pooled blood plasma study for volunteers from the blood bank where they looked at dioxin in the fat part of the blood. You can see that the numbers are fairly similar. Also, a study done by the National Institute for Occupational Safety and Health. These last two studies are not as comprehensive as NHATS, but I think they provide some confirmatory evidence.

Now in the second part of the table, the third and fourth lines, I have converted these into an estimated body burden. This was some work we did at the EPA panel. Essentially what you do is look at how much is in adipose tissue or in blood. You make an estimate about how much adipose tissue is in a human body and some other factors and you can come up with these numbers. You can see that, based on the NHATS data, which I think is probably about the best data we have, the body burden or total amount of dioxin-like compounds in Americans is on the order of 1.3 for TCDD alone, or on the order of 7.0 for TEQ.

You should also know that, clearly, these are average numbers and that there is a range of body burdens in the general population, where some people will be higher and some will be lower. It is difficult to estimate from the data presented in these studies what that range is, but as a very crude approximation, maximum concentrations could easily be two or three times higher. We don't really know from the data.

Now the NHATS data are very important because the way they were sampled was such that it was constructed to represent the average population in the U.S. That is very important and it has three additional conclusions that are of some interest. The first is that the concentrations increase with age. The second is that there was no significant difference by race or sex. The third, and probably most important, is that there is no significant difference among the four regions of the country that they looked at, except for one particular isomer, which was higher than average in the Northeast.

I am unaware of any currently available data on body burdens by the State of Maine. I understand there may be such work in progress now, but the data are not available yet and will not be for a while. So I think that in the meantime the NHATS data probably represents a reasonable estimate for the average body burden in the state.

One final thing about body burdens. It appears there may be a downward trend over time of the levels of dioxin-like compounds present in Americans. This is suggested by a comparison of the NHATS data results from 1982-1987 as well as a Veterans Administration analysis of stored body fat samples for the period 1971-1982. According to the authors of the report, this trend may reflect several factors. One is a true decline in body fat levels. The second is advances in analytical methods; that we've gotten better at detecting things and that can change the results. The third is that some of the tissue may be degrading over time in storage and that can change the concentrations as well. So while the data are not perfect in terms of what they are telling us, I think it is probable that at least some of that decline is real.

Now what should the Board make of that for purposes of risk assessment? I think, first of all, declines in body burden that are relatively small may still not provide a sufficient margin of safety. You can ask Dr. Hughes about that. Second, we cannot be sure that any past trend in decline in body fat levels will continue. For instance, although total PCB concentrations declined in Great Lakes fish during the 1970s, concentrations appear to have leveled off in the 1980s. I think this is something that was referred to obliquely earlier; that you can have a period of increased control over sources, which leads to a decline, but then things start to level off again. I think it is probable that we are seeing something like that in human adipose tissue concentrations of dioxin. Things that happened in the Seventies to control sources have led to a decline and things may be leveling off again now, although we don't really know for sure.

Now let me talk about background daily doses of dioxin. Remember that the average body burden for people in America and Maine is probably on the order of something like 7. Let's talk about where that material may be coming from. It is generally thought that the primary source--on the order of ninety percent--of exposure of humans to dioxin is food, especially fat-containing foods such as meat, dairy products, fish, poultry, and eggs. This is actually not very surprising. As you heard from Peter deFur, dioxin is persistent, it's fat-soluble, and it tends to bioaccumulate in the fatty parts of animals and fish. So it's not too surprising that that's the proximate source of where it's coming from.

This has an important implication. That is, a general contamination of our food supply may be accounting for the

similarity of body burdens between various regions of the country. So if the food supply is contaminated in some general sense, with some specific hot spots maybe downstream of a mill, the milk and meat grown in various areas but then distributed around the country may explain why the concentrations are as similar around the country as they appear to be.

One can estimate the average intake of dioxin from food by comparing measurements of the average concentrations of these chemicals in food with the aveage concentrations of those foods that people eat. That is called a marketbasket survey. You look at how much beef people eat, you try to estimate how much dioxin is in the beef, and you add all those things together and come up with a number. These sorts of surveys have not been done in the United States for dioxin-like compounds, unfortunately, but they have been done in several other industrialized countries, particularly Germany, The Netherlands, and Canada. They come up with numbers that are around 1-3 picograms/kg/day. In my opinion, although no complete marketbasket survey has been done for the U.S., it is reasonable to assume that those numbers will apply here as well.

Now with respect to TCDD alone, which is just one kind of dioxin, estimates of exposure in industrialized countries show an average dose of about 0.2-0.4 pg/kg/day. If you look at the levels of that one compound that are found in body fat, and take into account how persistent the chemical is in the body--which is around seven years for it to decrease by half--then, going from the body fat backwards, you can work out to how much people are being exposed to. When you do that exercise, in fact it comes out pretty close to what people have estimated for marketbasket. So that

gives us some confidence in that particular number and it also suggests that the current doses of TCDD are sufficient, or nearly sufficient, to support the current body burden of that compound.

Now I am unaware of any complete marketbasket survey looking at levels in food and how much food people eat--any estimate of that kind for total dioxin-like compounds in the U.S. or Maine. In its absence, I think the estimates from Canada and some of the other industrialized countries are reasonable proxies.

Let me summarize my two main points. I think that for an average body burden of dioxin and dioxin-like compounds in Maine, it is reasonable to use the national average for the U.S., which is 7.0 nanograms/kg toxic equivalents. Inclusion of dioxin-like PCBs would increase this value further. You should also know that some members of the general population, for a number of reasons, may actually have significantly higher body burdens. Second, a reasonable estimate of the average daily dose of dioxin and dioxin-like compounds for Maine is 1-3 pico-grams/kg/day of toxic equivalents.

Now let me refer you to the second table, which is at Tab 11 ["Proportion of Fish Dioxin-TEQ Dose to 'Background' TEQ Dose"]. This is an exercise that I've done specifically with some of the numbers you've heard earlier today. It is a calculation of the amount of dioxin that could be coming from fish in the State of Maine and comparing that with what I think is a reasonable estimate of the dose that we are getting from all sources. In the left-hand column, you have data on the toxic equivalent calculation--the amount of dioxin in fish--from different places in the state. For instance, the first line is the Maine average from 1992.

Then you have the various rivers. You can see that they go up to as high as 2.3 pg/g. That's the data that Peter Washburn referred to this morning. In the second column I put in some assumptions about the amount of fish people consume. These numbers come from Dr. Knuth's testimony. I have done some calculations here with three meals per month and four meals per month. Those correspond to 23 and 32 g/day. I have assumed a body weight of 60kg. Using that information, you can calculate the daily dose of dioxin-like compounds that are coming from the fish for a person who eats this level of fish. Those range from about 0.37 to a little over 2 pg/kg/day. So it's a fairly big range, depending on where people eat their fish from and how much fish they eat.

Now if you assume that the background daily dose of 1-3, which is reasonable for the industrialized countries, applies to Maine--and just for the sake of argument, take the middle value, which is 2, and you assume that's the total background dose people are getting, and you divide this estimated dose from fish into that, it gives you a crude approximation of what fraction of the background dose might be coming from fish in Maine under these assumptions. You can see that, in the lowest case, it was about 19 percent. That was for average Maine fish concentration and three meals per month. At the high end, about 63 percent, for the high concentration of 2.3 ppt and four meals per month. So that essentially means, if all these assumptions are correct, a little over sixty percent of that person's background dose would be coming from fish. I think that will come up again, but it's important to know that you can have a range of impact on the daily dose from 19 percent to a little over 60 percent.

STEVENS: Any questions?

ROY: The formulas that we've seen before have all consistently used a 70kg weight instead of the 60kg that you used as the weight of the average person. I wonder why there is that variation?

WEBSTER: Seventy kg is very often assumed for men and sixty kg for women. I think there is some pretty clear evidence that the fetus is the person we really need to worry about and they are exposed in utero. So this really is referring to a dose that the mother or mother-to-be- is getting. As you can tell, if you used 70 kg, it really wouldn't make much difference. It would only change the numbers slightly.

STICKNEY: Mr. Webster, you make the statement that all of us have dioxin in our systems--I guess unless you are a vegetarian--because we eat meat, eggs, dairy products, poultry, and some fish. You also made the statement that it has to do with fatty tissue. So would a thin person have less dioxin in their system than a person who is overweight?

WEBSTER: I don't really know that a lot of studies have looked at that. It is actually possible that a thin person might have higher concentrations because, if most of the dioxin goes into the fat, and there is less fat but they have the same intake, then they might have higher amounts in them. The body burden may not really be that different, but I am not really aware of a lot of studies that have looked at that. To respond to your other point, clearly, if you were a vegetarian, you might have lower doses. There are

some other sources of exposure besides food, but they are probably minor.

STICKNEY: On this last chart, you use the 1992 Maine average. When you say people are consuming fish at the rate of three meals per month, or maybe even four meals per month, is that considering all types of fish that people eat, or is that considering these four rivers in Maine that are the subject of our discussion that have pulp mills on them?

WEBSTER: I used the assumptions that were given to me by the other two speakers this morning. So I'm afraid you'll have to ask them as to exactly what is involved with the number of meals per month. What I asked them was, how much fish should I assume they eat. I calculated the impact of that. Perhaps someone else would like to speak to that question.

KREISMAN: Very clearly, the assumption here was that we were trying to show, if people were to eat fish from the affected rivers at the fish consumption rates which we believe are reasonable, what would we expect to find in terms of a daily dose coming in from dioxin. So you are exactly right, Mr. Stickney, but that was the intent of the exercise also.

STICKNEY: Because I have never eaten a fish out of any of the four rivers, and I eat a lot of fish.

KREISMAN: That might be. I think the surveys show there are other people who have.

STEVENS: I am extremely interested in the body burden from other sources, although dietary sources would include fish. Has this gone on long enough so that we have any indication as to whether that body burden--if half of it is being eliminated in seven years supposedly, so we must be adding to it all the time. Have we any indication of the stability of those figures? Has the study gone on for ten years, so that it is increasing, staying the same, or whatever?

WEBSTER: As I indicated in my testimony, there is some limited data on body burdens over time, from about 1970 through 1987. Although there are some problems with the data, it does look like there has been a gradual decline in body burden levels, which I think is consistent with some of the data on environmental contamination. For instance, if you look at the levels of dioxin in sediments in lakes--as was referred to earlier--there is almost nothing before about 1920-1930. Then it shoots up. Then it looks like there may be a decline from somewhere in the Seventies on, although it's kind of sparse information. So I think that a gradual decline in body burdens, at least so far, is consistent with some of the environmental data.

STEVENS: That gives us some reason for hope

WEBSTER: Yes, absolutely

STEVENS: Any other questions? ... Thank you very much

Dr. Claude Hughes for NRCM

I am Claude Hughes, currently a tenured associate professor in obstetrics and gynecology at Duke University Medical Center and a member of the integrated toxicology program faculty there. I have a bachelor's degree in biochemistry from East Carolina University, an M.D. and a Ph.D. from Duke in neuroendocrinology (which is how the brain controls reproduction). I completed a four-year residency in Ob/Gyn and am Board-certified in that specialty. I completed a clinical fellowship in reproductive endocrinology and infertility and am certified as a practitioner in that medical subspeciality.

I think is important for you to know that I did participate in the dioxin re-assessment at EPA's request. They asked me to be the principal reviewer of the chapter on reproduction and development. My task was to review that document and offer my professional assessment of the quality of the data addressed in that chapter and to provide an interpretation of the human health risks that might derive from those data.

When we talk about reproductive and developmental toxicity or toxicology, what I refer to isn't just whether animals continue making litters of babies when they are fed a particular compound, but other aspects of intact reproductive function and normal development. So toxic outcomes include disruption of normal processes in male or female animals or humans which are known to be essential for reproduction to occur and detrimental effects on the developing fetus which may manifest at birth or much later in life. This includes abnormalities of body systems;

not just malformations but other abnormal function of organ systems, including things as subtle as compromise of learning, alterations in behavior, even including sex-appropriate behaviors.

In the review for EPA, which was presented in open public meetings back in September, the injurious effects of dioxin on reproduction and development were analyzed. Various human and animal studies in which reproduction or developmental effects of dioxin were evaluated. We went through the additional exercise of comparing those effects to the amounts of dioxin and related chemicals which we currently bear--after the fashion that Tom Webster just described.

In addition to this sort of responsibility in the reassessment, I have authored or co-authored around ninety professional publications, about one-third of which are clinical--infertility, ovulation induction, human endocrinology type papers. The other two-thirds are basic science, which include effects of various chemicals on brain development, control of the pituitary gland, and reproduction. This includes an array of manmade and natural-occurring agents and drugs of abuse.

I have been a member of several committees and an expert on several panels, including food industry-sponsored participation on the International Life Science-Nutrition Foundation Expert Committee, which reviewed California's Proposition 65--with a rather critical view, I might add. I currently serve on the Board of Scientific Counselors for the National Toxicology Program, which is an over-arching program including portions of NIEHS, NIOSH, and the portion of FDA called the National Center for Toxicologic Research.

I also have a number of journal editorial appointments and referee services and so forth.

I think I can present you some fairly unique perspectives on these issues, given what I think are reasonable credentials, both as a toxicologist and as a practicing clinician with acknowledges expertise in reproduction.

Why am I here? I am here because the Natural Resources Council of Maine asked me. They are paying for my travel but no other compensation. So in fact my department is losing money by my being here. Why am I testifying? I think it is important. Having gone through this exercise with EPA, I feel that the people of Maine do face a real health concern regarding current exposure, current body burdens, current intake of dioxin and related compounds. I have real concern that intake of contaminated fish would incrementally add to that exposure and those hazards.

I base this perspective, as I alluded to before, on both my scientific training and interest as well as that of a practicing clinician who actually sees and takes care of sick people in clinic who come in complaining of reproductive problems, pregnancy loss problems, etc. Let me go through this in a more formal way. From that involvement with the EPA re-assessment, I'd like to offer a couple of conclusions. These are my own, derived from the review and thought processes that I engaged in.

There currently exist several reliable studies that demonstrate reproductive and developmental effects from dioxin in animals and people at relatively low levels of exposure; and when compared to the levels of dioxin that can be realistically expected to occur in people in Maine now, the levels in those studies correlate: the levels of effect

and the levels that we carry are rather similar. Thus, I have to conclude that there is a significant reproductive and developmental impact of dioxin and that this presents a significant public health concern.

Let me go into a little more detail. I think it is important for the Board to reflect on at least my understanding that the existing federal water quality standards for dioxin were developed before these developmental and reproductive issues were really brought to the fore. In 1990, when EPA decided to approve state dioxin water quality standards up to 1.2. ppq, the agency wasn't focusing on these things. But I can tell you that colleagues of mine at EPA and NIEHS were actively investigating these kinds of effects. EPA's current attention to these standards has been verified by a number of things they have done. They considered these kinds of toxicities of environmental pollutants to be important, as manifested by their dedication of entire half-days at this last review to reproductive and developmental effects and immuno-toxicity effects, which is another refreshing way to address these other kinds of health concerns that hasn't been done adequately in the past. Dr. William Farland made comments at the outset of that process and in letters communicating with potential attendees to the effect that all of these different issues were going to be taken quite seriously. That, too, was a break from past tradition of focusing on carcinogenesis only.

Another manifestation of that is that updated developmental toxicity guidelines did appear in the Federal Register last year. The amount of effort that went into those revised guidelines was substantial and is already serving an important role as guidelines for researchers in

academia, government, and industry for assessing developmental toxicity end-points or effects.

I have to say that I personally feel that several of these end-points may be more important than carcinogenesis, because when you talk about effects on the developing brain you are talking about the functional competency of the next generation. We are not talking about whether I get a cancer; we are talking about whether my children can perform up to snuff. That, I think, is actually more important. Now you have to think about developmental biology issues in a different way than if we talk about carcinogenesis, . This has been alluded to a couple of times. When you are concerned about development of the brain or other organ systems, the window in time during which critical events occur is in fact quite limited. Narrow time-limited exposures may have profound effects in terms of disrupting normal organization of tissues or systems within the body. Exposure to dioxin or other agents can have a profound effect over a short period of time. So an exposure in early or mid-pregnancy can indeed be transient but have permanent effects on the offspring.

This is a very different way of thinking as opposed to seventy years of ingesting anything to elicit a modest increase in cancer risk. These effects are not probabilistic but they are stochastic. So you don't talk about an occasional cancer victim occurring in a large exposed population. Rather, these types of developmental or neurodevelopmental effects are more homogeneously spread out across a whole population. Everybody suffers some, is a simplified way to describe it. This kind of effect, then, is more of a dose-proportional compromise of many members rather than one or two being singular victims.

As you consider any changes in regulation of dioxin, and perhaps creation of some interim standard, you have to keep in mind that it's very important to consider that limited exposures early in development, in utero or even in childhood, could profoundly affect the health of these humans permanently thereafter.

It is not possible to know at this time whether there are other periods in human life that are also exquisitely sensitive to perturbation, but things come to mind, such as the peripubertal interval. Again, for agents that are deposited in fat, during intervals of weight loss when you mobilize the fat, where does the dioxin go? It is into the rest of your body. Pregnant women commonly have an interval of anorexia early in pregnancy. That means they don't eat enough, they lose weight, they mobilize fat stores. Between thirty and fifty percent of women have some interval of anorexia. So we don't even know how high the peak may be when those women in the first trimester, especially late in the first trimester, are going through that kind of very common event where fat mobilizes. For any of us, especially as we age and have accumulating levels and we go on a weight-loss program, what happens to the dioxin in our fat? We have to mobilize it. I don't know of any data that assesses the changes in blood levels or other target tissue organ levels when that kind of phenomenon occurs. This is the fourth time I've brought this up in the last couple of months and no one can do more than shrug their shoulders and say, we don't know what happens. This, again, is talking about the existing body burden and the dynamics that may result from what we would construe to be positive lifestyle improvements. Getting yourself fit may not be a one hundred percent payoff.

Let me explain some further reasoning. Dioxin and related chemicals exert effects by binding to very specific nuclear receptors. These are similar to steroid hormone receptors. These are proteins that live in the nucleus of cells. For this family of receptors, there are 1-2,000 per cell. If you occupy a few hundred of those for other comparable nuclear receptor systems, you elicit biological effects, so you can occupy ten to seventy percent and you get different degrees of response by occupying those receptors. Everybody agrees that dioxin works by binding to these receptors. At a concentration of 1 ppq, a teaspoon of water contains over 1.6 million molecules of dioxin--compared to a couple of thousand receptors per cell. Of course, I can't say that all 1.6 million land in one single neuron in the brain, but this is the opposite of the stack of barrels to the moon. A very dilute solution contains a large number of dioxin molecules that are plenty to occupy the active receptors for these kinds of effects. This is very different from a compound like aspirin, which is a fairly general weak inhibitor of a generally present enzyme that involves synthesis of prostaglandins. Aspirin, which was mentioned yesterday, has not very specific effects compared to the "silver bullet" effect of these kinds of compounds.

As you've already heard today, the background level in humans for TCDD is something like 1.3 ng/kg, and for the sum total of toxic equivalents it is something like 7 ng/kg. Using either one of these figures, we now need to look at the studies that have been done in the last fifteen years that look at some developmental endpoints and some productive effects, different from the multi-generation, chronic exposure study that was mentioned yesterday.

In a NIOSH study, men who were occupationally exposed to dioxin showed suppression of testosterone levels. The body burdens in the men showing that suppression were in the range of 5 to >19 ng/kg. Even if we allow that we really can't determine whether the current body burdens or the exposure way back was the mechanistic cause for their reduced testosterone levels, effects of any of these numbers in humans is of concern to me. If these men came to clinic, they'd get treated for their lowered testosterone levels. They would elicit a health care response for hypogonadism.

You have to be critical about these things, no matter what interpretation you finally put on it. There is every reason to think that these men were exposed to other chemicals. But as the workers tried to look for other possible chemical mediators of this effect, they haven't found any. I know that is a double negative, but they can't really explain the effects based on the other known chemicals these workers were exposed to. Another point is that all of us are exposed to a whole array of chemicals. If there are adverse interactions among different classes of chemicals, we too are subject to those kinds of risk. It is a different kind of interactive concern.

Next, in a study by Mably and others, pregnant rats were given a single dose of dioxin on day 15 of pregnancy. The lowest dose studied was 64 ng/kg, compared to levels in us of around 7 ng/kg. That dose did not affect birth weights or adult weights of those offspring. It did alter male fetal development, such that they did have compromised sperm production, diminished size and weight of other hormone-dependent tissues like prostate and epididymis and so forth. So there were reproductive tract effects. And these males showed desmasculinization. So their behavior

was compromised. That's a brain effect. When these kinds of tests and basically any other exposure paradigm with any other agent have shown that kind of feminization or de-masculinization of behavior, subsequent studies in every instance have shown changes in brain anatomy and brain biochemistry that confirm that this is in fact a structural and/or biochemical change in how the brain works. How the brain is organized determines whether these behaviors are sex-appropriate or altered.

It is further important to consider that this was not a "no-effect level"; this was "low-effect level." To be sure we all agree on what that implies, it means that this effect at this dose might turn out to be microscopically above a no-effect level but it could be much higher. We can't tell until the next round of studies is done. And they are being done. So even if we accept that the low-effect level of 64 ng/kg is the level of dioxin which might turn out to have no effect on rats--which is a fairly significant and basically unfounded assumption--this would still be less than ten times higher than the body burdens we carry around right now. It is within an order of magnitude. So that is no margin of safety.

Third, in a study of monkeys by Bowman and others--which at one point was questioned about its reliability, but Linda Birnbaum and others at EPA say they have audited it and they are convinced that the study is clear--the offspring who were exposed in utero to 22 ng/kg showed changes in learning ability. They had disordered object learning. So in this primate model, another behavioral index of learning in the offspring was compromised by a dose that is only about three times higher than what we currently bear.

So why do I think that current levels of dioxin in people's bodies are a significant health concern, given that the levels I have described are at least somewhat higher than what we currently bear? None of these studies identified levels at which the reproductive or developmental effects do not occur. Therefore, we really don't know whether these levels truly are different than the dioxin levels that we currently bear. It concerns me that one of the studies indicates dioxin effects in humans at body burdens comparable to what people can be expected to presently bear. The Bowman studies in primates show effects on the ability to learn at levels only three times higher than what we all probably bear right now.

At this time there is very little information to make any judgment about whether humans are less or more sensitive to dioxin than animals, or whether or not wide differences in sensitivity to the effects of dioxin would occur within the human population. The only exercise that you can attempt is to look at the effects of dioxin in the occupationally exposed men and compare that to adult laboratory animals. In that comparison, it appears that humans are much more sensitive than the laboratory animals. That is the only best-guess comparison I can offer you.

Given these three sets of observations where the body burden in human study subjects or the animals are only three to nine times present human body burdens, I cannot conclude that there is any margin of safety between what all of us currently bear and the levels of dioxins and related compounds shown to produce adverse effects on male reproduction and central nervous system development, as manifested in sexual behavior and learning. The essence of these conclusions was presented at the re-assessment and

none of the assembled panel offered any real disagreement. The acknowledgment of that conclusion by EPA is described in the October 9th memo regarding the dioxin re-assessment activities that Erich Bretthauer, Assistant Administrator for Research and Development, sent to EPA Administrator Bill Reilly. So they acknowledged that interpretation and stated it, almost word for word, as I had stated it previously.

As a clinician, I can only see sustained risk if the levels of dioxin in the human body are allowed to remain elevated, and only increased risk if body burdens increase even incrementally. Let me give you some feel for the cost in human health impact on patients that I see. In terms of utilization of health care resources, infertility alone is a significant problem, independent of these neurodevelopmental type issues. Something like fifteen percent of couples in the U.S. suffer infertility. About forty percent of those are due to male factor. Most of those men have no obvious clinical cause for their low sperm count or low hormone levels and there is no clear history indicating some antecedent or ongoing injury. The clinical quandary is often trying to come up, for yourself and for the patient, with some cause-and-effect explanation. The only biologically plausible thing we are commonly left with is something environmental. I have no idea, for any individual patient, how you can indict any particular compound, except in certain instances where there is some industrial hygiene exposure study that has shown them to have been exposed. But one comes away with a sense that we don't have other biologically plausible explanations for most of these men's reproductive disorders. I have to wonder if, given what we now hear to be ancient history of dioxin and other compounds in our culture, dating from well before I was born, if many

men might not have suffered in utero exposure, even decades ago, and now be suffering long-term consequences. That is biologically plausible also.

Another point regarding women eating fish. More than fifty percent of all human pregnancies in the U.S. are unplanned. So when you talk about protecting the fetus, a couple can't be expected to plan ahead and say, we're going to eat right and do all the right things to protect our future baby by changing something over the short term, because that will only address risk reduction or improved quality of lifestyle for maybe 45 percent of all pregnancies. If you talk about exposures that have a timespan of many years, then we would be demanding outrageous things: a teenager planning a pregnancy twelve-fifteen years later, to change her lifestyle to reduce fetal exposure. That's impossible.

On an individual level, when patients show up with abnormal sperm counts, disorders of ovulation, changes in menstrual function, etc., they have to be treated. Men with low sperm counts will come to the clinic for infertility assessment. You do a number of screening studies and commonly end up only being able to offer high-technology options like in vitro fertilization. Many of them will conceive with those kinds of technology--at $7000 per treatment cycle and a 1-in-10 pregnancy rate per treatment cycle. There is now internationally based data to say that four or five cycles is statistically reasonable for those couples. That's many thousands of dollars to treat one couple for a treatable problem. So there are very real costs of even the simplest case of compromise of these individuals. The bottom line is that such patients with diminished sperm count or lowered testosterone levels do

incur very real costs in medical treatment. Frankly, I think it is ethical to say that any preventable cause for such disorders is worth addressing from both public health and health care financing perspectives.

It is not as easy to measure changes in sexual behavior as would be implied by some animal studies, or learning disorders as implied in the monkey study, but I find those particularly worrisome--as a physician, a parent of two little children, and as a citizen with some concern about the functional status of the next generation. As a scientist in reproductive developmental toxicology, I believe that the available studies show the hazards from dioxin and related compounds can occur at levels that are very likely in the range of present human body burdens. As a physician, I can't think of a justification for any collective behaviors that serve to sustain a current hazard or permit it to worsen.

In closing, I'd like to leave you with these two opinions: (1) Because existing levels of dioxin in people's bodies are of significant reproductive and developmental concern, regulatory efforts should be focused on severely reducing, if not eliminating, sources that create body burdens of this group of chemicals. (2) To the extent that the consumption of dioxin-contaminated fish in Maine rivers has an effect of either sustaining or increasing those levels, then I think that is a significant public health concern.

STEVENS: Any questions?

STICKNEY: You mentioned contaminated fish, but the previous speaker, Mr. Webster, spoke of dioxin as being in meat,

eggs, dairy products, poultry, and fish. So can you tell us a little about the danger all of us are in who eat those products but do not eat the fish out of these four rivers that are under consideration in this hearing?

HUGHES: What is reasonable to suppose is that all those things contribute to what we bear. I think what makes public health sense is to try to reduce all of those exposures as much as we can. It may be a necessary exercise to fragment those efforts to reduce exposures to these kinds of compounds into different topics for regulatory purposes. You know more about that than I do. But issues about incinerator sources, smelters, etc., I think are also important and need to be addressed. I don't think it's one or the other; I think it's all of the above. As one who grew up on a dairy farm, it concerns me that dairy products are another source of this.

STICKNEY: Do you have knowledge that the EPA is pressing down on the cattle industry, the pork industry, the lamb industry, and all the other things that were mentioned? Are they coming up with the same type of scrutiny as the dioxin coming from paper mills?

HUGHES: I haven't heard of such. As a member of the Sheep Producers Association of the country, I figure I'd hear about it pretty early.

STEVENS: See if I have gained the right knowledge from your very interesting testimony. Is it fair to say that your major concern is not so much risk assessment of the carcinogenic effect of this compound and spending major time

on that, but really what we should be more concerned about is the hormonal receptors located in our cell nuclei that may be taking up dioxin or its allied compounds, and thereby preventing proper hormonal contact within ourselves?

HUGHES: Bingo! You've hit it on the head. When the scientific data base gets sound enough, as it is rapidly getting with dioxin-related compounds, you then understand the molecular and cellular mechanisms of action. It looks like these divergent effects on reproduction, CNS function, immune function, hepatic (liver) enzyme changes, carcinogenesis--all appear to be unified by an AH receptor mechanism. So once you gain enough data to make that mechanistic argument, then I think many things become clearer. One can then look for those cellular markers in fetal tissues and get a much clearer meaure of how much the hazard is for target tissues by using animal models, etc. That is very satisfying scientifically and should allow the scientific community at large to give you better advice and say we are really confident because we finally understand how it happens. For a number of target tissue effects, I think we will have that kind of understanding before we understand carcinogenesis.

STEVENS: Thank you. Any other questions? ...

Dr. Ellen Silbergeld

You have a résumé of my background and experience and I'll only touch on those that I think may be relevant to your judgment of what I would like to present to you today.

My education and training has been in environmental engineering and in environmental toxicology as well as in basic research focused primarily on neurodevelopmental toxicology. I am currently a tenured full professor in epidemiology and toxicology at the University of Maryland Medical School and an adjunct full professor of environmental health sciences at the Johns Hopkins medical institutions, both in Baltimore. I am also a senior adjunct scientist with the Environmental Defense Fund with my colleague, Dr. Peter deFur.

My research experience over the past twenty years has resulted in publication of about twenty papers, book chapters, and abstracts, primarily focused in the area of neurosciences and toxicology. I have been directly involved in conducting basic research on the toxicology and mechanisms of dioxin since 1982. I may be the only person who has spoken to you who has actually handled and dealt with dioxin in the laboratory.

I have served on numerous advisory committees related to toxicologic matters, including serving as a special consultant to the U.S. Secret Service, to the Organization for Economic Cooperation and Development, New York State, the Government of Bermuda, the Centers for Disease Control, and the World Health Organization. I have served specifically on dioxin-related committees for the National Academy of Sciences and also on committees related to risk assessment for the National Academy of Sciences and am currently a consultant to the Institute of Medicine Committee on Agent Orange. With respect to Agent Orange, I am a member of the science advisory committee to the American Legion and also to the U.S. government for its studies of Air Force personnel exposed to Agent Orange.

I have served on the EPA Science Advisory Board executive committee and have been an appointed member of special advisory committees to EPA in 1985, 1988, and currently for the various assessments and re-assessments of the toxicology and risk assessment for dioxins and related compounds. In addition to serving on the Science Advisory Board itself for EPA, I have served on the National Academy of Sciences' Board of Environmental Sciences and Toxicology and I am currently, with Dr. Hughes, a member of the Board of Scientific Counsellors for the National Toxicology Program of the United States, and a member also of the committee that reviews data on the scientific evidence for carcinogenicity of chemicals and other materials. Like Dr. Hughes, I review papers and grants. I am on the editorial board of a number of journals. I am past president of the Society of Occupational and Environmental Health.

I would like to speak with you on some of the matters that have already been raised and try to bring them together in a way that may make sense. I would like, however, at the outset to invert the commentary of Admiral Stockdale and say: why are <u>you</u> here? You are here because the EPA is not here. You have been placed in a very difficult position, which is to examine an extremely complex set of information related to chemistry, biochemistry, biology, eco-toxicology, ecosystems biology, fish consumption, human behavior, industry policy, and economics. One of your duties must be to keep those issues separate and to determine, the best way you can, to bring to bear the most relevant and directed state-of-the-art consensus scientific information that coincides with your mission to protect human health and the environment through the application and enforcement of rationally based water quality criteria. All the other

issues that relate to those must, in my opinion, be informed by a primary judgment as to the potential hazards of various standards for dioxins in receiving waters on human health and environmental integrity.

Now when we approach this overall assesment from that perspective, I would like to state what I believe to be the scientific consensus about the hazards and mechanisms of action of dioxin. I will not make reference to the work of the re-assessment committee, although I am an author of the final summary chapter, which is the dose-response chapter. I will be happy to answer your questions about that document and the views and opinions of my colleagues in the preparation of that entire re-assessment. I want to make it clear that I am not trying to tell tales out of school or preëmpt the publication of that work, but to present to you what has been the result of decades of research by myself and others on this topic and relevant areas.

It is very important now, in 1992, to acknowledge that we have very solid information on the identification of the hazards of dioxins and related compounds; that is, the biochemical properties of this extraordinary molecule and its related structures. It can be best described--and I think Dr. Hughes led up to this--as an extremely stable synthetic hormone. So imagine the dispersive release of an extremely stable synthetic hormone into the environment. I think Dr. Hughes was also very right to finally confront this continued repetition of drops of water in swimming pools, pennies on the way around the earth, barrels on a trip to the moon. This is an absurd way to understand the way in which hormones act in the body. Hormones are very different. They are, as he suggests, a "silver bullet." They have an extraordinary biologically based tracking

device, so that, unlike the one barrel in the millions on the way to the moon, they are in fact that one barrel that lights up when the eye of the cell, the receptor in the nucleus of a cell, scans all the molecules presented to it. All that it sees is that one molecule that fits like a key into the biological lock, which is then opened on a structurally specific basis by hormones, of which dioxin must be considered one. So I hope that lays to rest finally all this gibberish about how insignificant and silly it is to talk about parts per billion and parts per trillion. It is not silly to talk about ppb and ppt when we talk about steroid hormone action. That is what we are about here today.

The hazards that are now well associated with dioxin are the hazards of cancer, reproductive and developmental toxicity, immune suppression, and neurologic damage. In addition, there are target organ effects on liver, skin, and kidneys. These effects can be grouped in terms of the consequences of chronic exposures, of which probably the most important are cancer and hepato-toxicity (effects on the liver) and, very importantly, the consequences of much more limited or short-term exposures. I would like to focus on the latter, because of your concern about the potentially limited nature of the consequences of your decision here, although I would note that that is potential. The actual duration of the consequences of this ruling are, I think, somewhat uncertain.

As Dr. Hughes mentioned, there are at least three systems where the biology of the system is such that very short-term interventions in its status have long-term, if not permanent, consequences. These are systems that are acutely attuned to developmental state of the organism.

They undergo a very precisely timed developmental acquisition of competence. In the case of certain parts of the reproductive system and of the nervous system, there is no opportunity to recapitulate, to remake the system once it has gone down a certain pattern, be it the biologically corre correct one or a biologically deranged one. We don't a chance to repair these systems.

The three are the immune system, the reproductive system, and the nervous system. The reproductive system and the nervous system, as I mentioned, have exquisite timing which cannot be recapitulated, cannot be repeated. We know this from many agents that damage these systems. We know that early, highly limited exposures--prenatal alcohol syndrome, early lead exposure, early drug exposure--have profound and, as far as we know, persistent effects on the later acquisition of full developmental competence in both nervous system function and reproductive system function.

The immune system is even more developmentally sensitive in that, at very precisely timed periods in the late prenatal and early postnatal periods, various parts of the immune system come into full function and, very importantly, communicate with each other in order to acquire that full functioning. Therefore, very limited perturbations in that system, depending upon the time that they occur, can have devastating effects on the acquisition and maintenance of immune competence. We know that now, too, from a number of drug studies as well as inborn errors in immune system function.

These are the systems that are very sensitive to the effects of dioxin and they are sensitive precisely timed with the periods that are most critical to the later development of full competence. So this interaction between

ontogeny, or the development of the systems, and the sensitivity to dioxin is not coincidental. Nor is it coincidental, I think, to a relevance to cancer, as Dr. Hughes mentioned, because we are now looking at a fundamental set of mechanisms that play out in different cells--perhaps even in men and women and certainly in different age groups--depending upon the demands on those systems, other endocrine and hormonal changes that are ongoing, and the life history of that cell and the function that is sensitive to dioxin at the time.

Dioxin is clearly the most toxic manmade chemical that we have ever studied, other than lethality. Its reputation as being acutely lethal has been something of a diversion. There are a number of biological molecules that are equally lethal. But it is not weirdly toxic. It's not an outlyer in our scientific knowledge. I'd just like to stress again that its potency very much resembles that of endogenous steroid hormones. It is reminiscent of those molecules, both in how it acts as well as in its potency.

It is the scientific consensus on the mechanisms--the topic that Dr. Hughes ended with--that I think is important for you to consider, although not necessarily to apprehend and utilize as a critical component of your decisionmaking. I would like to stress that this information is a consensus opinion. This is not an outlying or vanguard notion in our understanding of the biology of the mechanisms of dioxin. This has been the focus of my own research and it is research that has grown and developed over the past twenty years.

We understand now that the biologic basis, the initial event, upon which all the cellular and organ and organism level effects of dioxin appear to depend is in fact inter-

action with a nuclear steroid hormone-like receptor, which has in fact been called the dioxin receptor. The property of that endogenous molecule, which is found in human cells as well as in the cells of sensitive animal species, is to recognize and bind to dioxin, transport it to the nucleus, and facilitate its interaction with specific genes. Thus, dioxin is a genotoxin; not in the sense that it mutates genes but in the sense that it profoundly alters the expression of genes. The analogy would be, if you had a Xerox machine to make copies--which is essentially what genetic machinery does--you have a piece of paper which has the instructions for your cell in your genes. But in order for something to happen, you have to make a copy and take it out so that something else does something in response to that message you take. Now you can damage this process by either messing up the Xerox machine itself, by ripping the copy after you've made it, or by interfering between the piece of paper you're trying to copy and the copying mechanism. Essentially, dioxin does the latter. It interferes with the expression of specific target genes. As a result, it deprograms and reprograms cells.

Now this occurs at molecular concentrations, as Dr. Hughes mentioned. These millions of molecules of dioxin contained in a teaspoonful of water, with parts-per-quadrillion (ppq) concentration, are highly relevant to your evaluation of hazards, because you are talking about confronting an exquisitely sensitive, evolutionarily tuned mechanism--overwhelming it with molecular confrontation with highly stable molecules. The difference between dioxin and endogenous steroid hormones is that it is extremely difficult for the cell to get rid of dioxin. Its stability and persistence aggravate and amplify the biological signals

that it induces through altering gene transcription. That persistence, which plays out on a macro-scale as Dr. deFur has pointed out in terms of its behavior and penetration throughout ecosystems and up the food chain to top-level consumers such as ourselves, is also played out in the micro-scale within cells. That persistence is one of the critical characteristics of these compounds.

A receptor-based approach to assessing the risks of dioxin, which is the consensus approach based upon the consensus statement of molecular biology of this compound, is in fact the sound science upon which criteria for understanding and assessing risk will be based in the future. This consensus, I'd suggest to you, allows us to understand the multiplicity of effects, including the reproductive effects, of dioxin.

I'd like to turn now to some of the implications of this understanding of the risks of dioxin and the way in which they affect. Dr. Hughes pointed out one of the important differences between reproductive toxicity and carcinogenicity in terms of a kind of bottom line. Are we concerned about an increase of 1-in-1,000,000? And there may not be a million people in Maine who are eating fish from the Androscoggin, and therefore would we ever see anything really? Or are we concerned about a different kind of risk altogether?

Using the overhead, I'd like to show you conceptually what I think you need to think about here, because these are conceptually very different risks. As a consequence, there is a very different bottom line you need to think about. This really just amplifies what Dr. Hughes mentioned before. We are essentially talking about two different kinds of effects that have very different public health consequences

that you need to think about. The kind of effect we usually think about when we talk about risk assessment is, looking at a dose, as it increases, a risk increases. You've heard a lot about those risks. They are always expressed probabilistically: 1-in-1,000,000, 1-in-100,000, 1-in-10,000. At one dose, let's say, there is an associated risk of 1-in-1,000. Obviously, none of us would tolerate those kinds of increases in risks in considerations of this sort. Then, at some higher dose, there is a higher risk of, let us say, 1-in-100--very high risks. And so we would talk about a risk assessment curve of this nature. We might assume it is linear or non-linear. Let's assume it is linear for simplicity's sake. But one of the important aspects of this kind of risk, as some of you have noted, is that actually, if we take it very crudely, 999 times out of 1000 there is no risk. That's what we mean by probability. So, all things being equal in a very simple world, for 1000 people equally exposed, with equal susceptibilities and histories, one of them might experience a cancer associated with the exposure, but 999 will not. Even up here at 1-in-100, 99 times out of 100 nothing will happen. So these are the dimensions in which you can choose to look at a kind of bottom-line approach to probabilistic outcomes like cancer. After all, cancer--like pregnancy--is something you either have or you don't. You don't have a little bit. So that's why it is expressed in these probability terms. It's like flipping a coin: it's either heads or tails; it's not a little bit of both--unless something has gone very wrong in Atlantic City.

Now I'd like you to contrast that with what we know about reproductive and developmental toxins--just in terms of the bottom line. Let me draw a real dose-response curve

that was published in The New England Journal of Medicine last week, related to lead--a very well characterized neuro-developmental toxin. Now we know that, as the dose of lead goes up, we can look at an effect. The effect that was studied in this particular paper was the IQ of children. A very important behavioral output, we'd all agree, of our children--their intelligence. What this study, and other studies as well, show is that, as blood lead levels in children go from 10 to 30, the IQ drops about 15 points. But that's in every child. That's the difference. So although we may say a decrease in IQ is not as serious as contracting a lethal cancer, I think the point that Dr. Hughes was trying to make is that the prevalence is so much greater that, for continuous effects of this type, where the effect severity, and not the probability, varies with dose, the public health bottom line may be extremely serious. These sorts of effects, therefore, deserve your attention to a very great degree in proceedings of this type.

Now I'd like to end by returning to the issue of how one can scope the data you have related to exposures and actual concentrations and events in Maine with the type of toxicologic and basic research data we are trying to present to you. I think a very convincing case has been made to you, and has been made in many other fora around the world considering this issue--at the World Health Organization, the Centers for Disease Control, the EPA, and elsewhere--that background levels of exposure and body burden in many industrial populations, including our own, are in a range of concern. For particular populations, such as pregnant women and nursing infants, they are possibly within the range of actual toxicity, particularly for neurodevelopmental, reproductive, and immunologic effects. So that our goal

must be to look at this as a need to reduce the background; not to deal with the additive exposures upon the background.

You are confronted with a very difficult situation. As many of you have already pointed out, there are multiple sources. In fact, there are not only multiple sources right now; there is the burden of the past overlaid on ongoing sources. So what is a reasonable decision in confronting one source, which is the power you have before you right now, within this mosaic of multiple past and present inputs into the human daily dose and the human body burden? I'd suggest (with humility because it's not my field) that obviously one should take into account such reasonable principles as cost-effectiveness and where you can make the best investment. The best investment is clearly in the food chain and in modifying consumption and exposure to dioxin that occurs through ingestion of food, because we understand that is the source for most people of the major part of the daily dose.

Now what is the most efficient way to do that? I do not think it is to go after the milk producers, the lamb and veal producers of the United States, because that is only secondary. There we are interdicting the outputs. It is much more efficient and reliable to interdict the inputs. Where are lamb, sheep, milk, eggs, poultry, fish getting the dioxin from? In this case, we know where the fish are getting the dioxin from. So all standards of engineering and economics and rational government behavior, or least intervention, say it is to interdict the primary input. You have a remarkable opportunity to do that, to take a reasonable exercise in pollution prevention to stop the accumulation of this process through a food chain that

eventually leads to top-feeders in our ecosystem and wildlife populations and humans as well.

There is a direct analogy with lead in that the most effective thing we've ever done in the environment for public health is to remove lead from gasoline. That was an interdiction at the point of input; not at the various outputs.

I think this can in fact make a real difference. Let's look, for instance, at the data that Tom Webster showed you of the daily dose, the proportion of the fish TEQ dose to the background TEQ dose (at Tab 11). What these data impress upon me is that, for someone eating three or four meals a month from fish with these representative levels in these rivers, that consumption pattern constitutes a significant portion of that individual's background dose. So this is not a trivial part of this complex picture of exposures to focus upon. This is a substantial portion. Let me remind you that the Public Health Service congratulates itself (with good reason, in my opinion) that taking lead out of gasoline reduced human body burdens by about thirty percent. You have a potential opportunity to do the same thing here by focusing on this one input.

Now let us look at these data in another way; that is, in terms of the daily dose and the range of options that you have before you. The figure at Tab 13 is an attempt to draw some implications for the daily dose from a variety of water quality standards that you might consider. If you consider the current discharges--whatever those might be--as being related to dioxin concentrations in fish of approximately one ng/kg, that then yields (and Mr. Webster went through this) a range of possible daily doses of 0.36 to 0.54 ng/kg/day (the middle column of figures). That, as noted

before, is between twenty and thirty percent of the background intake dose--a not-insignificant part of the overall picture to deal with. If you were to go to the proposal of 0.5, and if in fact discharges went to that level, the fish dioxin concentrations could range between 1.3 and 7.1 ng/kg. Again, a scenario of three or four fish meals [per month] of such fish would then present daily doses as shown. That would then be a substantial amount of the background dose--from 25 percent to an almost doubling of the background dose. That is certainly not insignificant.

But what is the advantage of undertaking and imposing upon the people of Maine the standard of 0.013? What are you going to get out of this investment? Here is where I think these data are very worth your consideration. That is estimated to eventually reduce fish dioxin concentrations to about 0.2 ng/kg, with a resultant reduction in the daily dose, as shown in the last lines of this table. That will then reduce this one source for the overall daily dose to 4-6 percent. I'd suggest that, from the public health point of view, if we could find one step that would make that kind of impact on an exposure to an identified hazard, we would be very glad to take that step. And compelling evidence would have to be presented against taking that step.

STEVENS: Thank you very much. I have two questions. If we go back to the receptor theory, does that give us comfort, or is it a possibility that, because this receptor is there ready to accept the dioxin molecule, a single toxic dose during pregnancy (or whenever it occurs), once it comes in and reaches the toxic level and is taken up by the receptors, that's all that's necessary? Then it remains

there and exerts its influence from then on? Is that a possibility?

SILBERGELD: Yes, it is. It may be a result, either as you suggest, because the dioxin itself stays there for some period of time or because of the critical timing at which that event occurs. We have an analogous chemical that has caused devastating health effects in our population. It is called diethylstilbesterol. We have experience with what happens with exposures to synthetic hormones at critical periods of development.

STEVENS: To the next generation?

SILBERGELD: To the next generation and potentially the generation after that as well, I believe.

STEVENS: The other question you alluded to, you said you were not going to mention this unless asked. Since you are on the panel that is seeking to establish a federal level, can you enlighten us as to whether you think, because of the reproductive possibilities, it will be the same as the standard now, or lower, or what is your thought on that--if you want to answer. You just sort of alluded to that.

SILBERGELD: I am one of the co-authors of the final integrating chapter of the re-assessment volume, the chapter on dose-response. One of our central tasks is to provide for the agency a biologically based model for risk assesment for the dioxins, out of which the agency can then make a policy decision as to where on those dose-response curves it wishes to align itself and make national investments. But

the shape and nature of that dose-response curve is what we have been charged with developing in terms of the science. For cancer alone, the risks of dioxin will only be considered more serious.

STEVENS: Thank you. Anyone else have questions?

ROY: You indicated at the outset of your testimony that yours was what you called a "consensus opinion." I am frankly having a very difficult time squaring that with industry's position. Are industry's arguments regarding the clinical effects of dioxin really on the fringe? I would like some sort of response to that.

SILBERGELD: I wasn't here during their presentation, so if there are specific issues that you want to ask me about, I'd be happy to try to respond. I would only note that there are a lot of people who have opinions about dioxin, but there are very few us of who actually work with dioxin. I am speaking of the consensus opinion of researchers actually involved in the epidemiologic and toxicologic studies of dioxin. There are scientists who work for the tobacco industry who say that cigarette smoking doesn't cause cancer. You can judge where you think they lie on the consensus spectrum. But if there are specific statements that have been made here before you, I'd be happy to try to respond to them.

ROY: One of the statements you've already spoken to--the issue of the teaspoon. It's very difficult for me to get any sense of what is real. You've certainly added to that

but you are one person and we have to make some judgments based on an awful lot of things.

SILBERGELD: I'd say with some degree of confidence that if you asked the larger community of persons expert in steroid hormone biochemistry and physiology how they would try to present to you the extreme potency and sensitivity and specificity of the action of steroid hormones, they would use the kind of language that Claude and I have used; that it's better to talk about a searchlight picking out the one spot in a universe of darkness than one drop in an undifferentiated swimming pool, which is the analogy that has too often been used.

STICKNEY: I must come back to the statement by Mr. Campbell that we are all at risk in this room because I think all of us eat eggs, drink dairy products, meats, and what have you. Very few people in the State of Maine are subjected to even having the opportunity to eat one of these supposedly contaminated fish from the four rivers in which pulp mills exhaust their effluent. So I guess I am more concerned as an individual from these talks this afternoon with what risk am I at and my children and my family from eating beef or lamb or the other products that are also carrying dioxin. I know that is not the subject we are discussing here, but it has been brought up and used as part of the dioxin chain or source, so I think all of us should be concerned as to what the future poses for all of us in eating those products.

SILBERGELD: I think that's true. I think that's why a comprehensive view on exposures, which I understand is

another aspect of the re-assessment that EPA is undertaking, needs to be done. What I suggested at the end was that you take a very hard-headed view and say that, in that context with many sources, many different diets and lifestyles, what is the worth of an investment in this particular source. I'd suggest that it's sufficient to merit your investment. It's not the end of the problem in the same way that taking lead out of gasoline didn't end lead poisoning. But it made a dent that was noticed.

TRACY: You've been very enlightening and I have to echo other Board members that the data is, no question, over-whelming in trying to take it all in and put it together and make some sense. My husband is an avid fisherman and hunter. He has a thirty-pound lake trout on his wall to verify that. However, it comes from Canada and not Maine. I am sitting here as a mother of two teen-age boys who are also fishing and hunting. Quite frankly, the meat that we have either comes from the one moose catch or the yearly deer catch. I am thinking that the dioxin level is not just in the rivers, as you are are indicating. Even though the moose may have come from the Moosehead area of Maine, there is still dioxin there. And it is not necessarily from the paper industry. The whole emphasis for our being here is based on the paper industry. However, what has come out mainly today is that dioxin is dioxin is dioxin--whether it comes from the paper industry or whether it comes from the air or Agent Orange, it is there, we are getting it every-where.

SILBERGELD: But not equally from everywhere. I really would urge you to look at the table in which we tried to

draw for you the implications of different water quality standards for the daily dose. Just consider the request of the Natural Resources Council--which I know they took with a great deal of debate because they called me repeatedly late at night about it--and I am very impressed with that, too, I must say, and I am here without getting any money for this either, except my plane fare, I hope. The ability to reduce an identifiable portion of the background dose by about fivefold is very significant from a public health point of view. That's not an insignificant opportunity you have before you.

TRACY: You are zeroing in on what is before us, which is the rivers of Maine. I certainly understand that argument. I think, however, especially the past two days, the emphasis and the presentations have been over the entire gamut of dioxin. I understand that that was done on an educational basis for us to understand the severeness of how dioxin affects us as human beings. There would also be the argument--and we've heard it today--that we don't fish the rivers, or we don't eat the fish from the rivers. As avid fishermen as my husband and two sons are, the fish that we do eat would have to be at least ninety percent from the fish market, which would not come from any of the rivers in Maine. Leaving here today and hearing the general public who have read the articles, who have seen the televised as well as radio coverage, they would say, I am not in that population, what's the big to-do? Although we are going to hear, I am sure, from the Native Americans that, yes, they are in that population. How do you counteract all of this?

SILBERGELD: That is certainly an option. We can take a quarantine approach to the environment. We can dump toxic chemicals in certain areas and we can post them. We can fence them. We can put guards with guns around them. I don't know how much of Maine's economy depends upon tourism, upon the love of people like me who grew up in this region wanting to come back. One of the things we do like to do is fish. I like to fly fish. I don't think I want to go to a state where I get the concept that rivers are being "let go" and that I just get a map from your fish-and-game people that says, just don't go here and everything will be fine. It would give me a kind of uneasy feeling.

I think there are commitments and fundamental values at stake here as to how we treat certain parts of our population and how we treat the environment. Do we treat it as something that is quarantinable? And that's how we handle these problems? Or, if we have an opportunity to intervene and make a difference, do we take that opportunity? I think that our laws and our ethical tradition incline us toward one direction. And I hope we are not going to change that.

TRACY: One more question. In another debate that took place three days ago, the argument is the economics of it--the fact that we are going to lose out in competition. Our competitors are not under the stringent regulations that our industries in Maine are. You are co-authoring a document that hopefully will be passed by EPA. I asked this a day or so ago: What are the chances that that document will be accepted and that all states within the United States will be treated fairly as far as these levels?

SILBERGELD: I guess I'd say I think the chances have changed considerably as of two days ago. What you've spoken to is a way in which the war between the private sector and government has led almost to quarantining of certain states in which they've bought off on allowing themselves to be degraded and placed in a terrible vise between economic development and public health, which is intolerable. I think, if I may venture to say, that we are certainly at an interim point. You are at an interim point in the official scientific assessments of dioxin. You are at an interim point in the actions of official agencies. But you are also at an interim point in the technology of paper and pulp production. Many other countries are going to chlorine-free methodologies for a variety of reasons. In fact, some of the analyses that have been done in Sweden and in Japan suggest considerable cost savings of making that investment at this point. If you are going to take an economic look at this issue, you should take a comprehensive one.

TRACY: The only other concern, based on what Ron Kreisman submitted this morning, is that Arkansas and Tennessee have worse standards that we do in reference to this. This kind of shakes me up a little bit. Thank you very much.

STEVENS: Thank you

Closing Statement by Ron Kreisman

We are asking four things from this Board: (1) We are asking you to undertake and follow your legal responsibility of protecting the most sensitive designated use of fish for human consumption in establishing, or not establishing, a rule. (2) We are formally asking the Board not to take any action that would keep fish contamination at its current level or make the situation worse, either by adopting this rule or by adopting, under the other portions of the toxics rules, a 10^{-5} level which would immediately apply to dioxin. We are asking you formally not to undermine the actions of EPA, because adopting this rule will keep the rivers off-limits for the length of this rule and its effect. (3) Personally, I am asking you to decide as you are driving home tonight to ponder whether you would be prepared, knowing what you heard today, to feed or to continue to feed fish from Jay, from Lincoln, from Augusta to a pregnant daughter, to a pregnant friend. If you are willing to do it with a clear conscience, you might want to enact this rule. But if you twitch, if you twitch, you can't pass this rule, because you are willing to pass sentence on other people and essentially quarantine the environment.

Finally and most profoundly, (4) when you address this very difficult issue--not black or white--whatever the Board does, NRCM implores you to be honest and straightforward with the public in what you decide to do. If you decide that, for the economic reasons you've all heard, this water quality standard should go forward, say it. Say it publicly, say it to the Legislature that you are making this choice, and say it so we are sure fish advisories are going to stay on these rivers. Let's make the choice clear.

Let's not weaken the water quality standard because public health allows it to be weakened, but let's make the choice very clear. It is a legitimate public policy choice, as Dr. Silbergeld indicated, to say that the cost of clean-up is not worth the benefit to the Penobscot Nation or whomever. Maybe that choice should be made in the Legislature and not before this Board under your statutory mandate. It is a legitimate choice, but it is not legitimate to do it under the table without telling everybody what you're doing, because if you do and change the water quality standard, as night proceeds into day, those fish advisories will be lifted. That will be the result.

STEVENS: Thank you. We will take a brief recess before hearing from representatives of the Penobscot Nation.